

## **At the Intersection of Public Health and Criminal Justice Research on Drugs and Crime**

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### **Introduction**

This paper discusses intersections of public health research and criminal justice research on the topic of drugs and crime. The drugs of interest mainly are marijuana, heroin, and other internationally regulated compounds of illegal origin, and such internationally regulated products of legal origin as pharmaceutical cocaine hydrochloride, codeine, and oxycodone, which also may be consumed on an extraordinary basis (i.e., outside the bounds of accepted medical practice). An important point of departure for this paper is a widely held assumption about two goals of research on this topic. The first goal is to achieve greater understanding and develop a body of definitive evidence on drugs and crime. The second goal is to achieve greater mastery of the design and application of policies, programs, and techniques to improve public health and public safety by preventing and reducing harmful consequences of drug use.

The outline for this paper corresponds with assignments delegated at a planning meeting held at the National Institute of Justice (NIJ) in January 2001. This introductory section provides some background notes on the literature reviewed for the paper and describes an organizing conceptual framework that can be used to assess gaps in the current evidence. The next section identifies some tensions that merit discussion as we try to forge a new research agenda on drugs and crime. We then address the central question posed in our planning meeting for the drugs-crime research forum: “What do we know about the drugs-crime interrelationship?” We cannot provide a comprehensive answer to this question in a relatively short paper, but we will offer a starting point for discussion, focusing on suspected causal relationships between drugs and crime. We also present a few concluding statements that were designed to facilitate discussion at the forum on drugs-crime research held at NIJ in April 2001.

### **A burgeoning literature on a variety of fronts**

A scholar interested in the topic of drugs and crime has much to read. Some of the classics of the field include Terry and Pellens’s *The Opium Problem* (1928); early papers on drug taking and sociopathy by Kolb and Pescor, who were two of the early clinical leaders in research at the facility that ultimately became the National Institute on Drug Abuse’s (NIDA’s) Intramural Research Program and Addiction Research Center; and work by Dunham and Lindesmith, whose surprisingly contemporary remarks and

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observations started to systematize some of the field's research questions on the social psychology of the drugs-crime relationship. Many of the issues that confront the drugs-crime researcher today were articulated by Terry and Pellens (1928), Kolb (1925), Pescor (1939), Dunham (Faris and Dunham, 1939), Lindesmith (1938), and their contemporaries in the first half of the 20th century.

These issues were re-articulated and a new set of themes was clarified in subsequent research, such as *The Road to H* investigations led by Chein (1964), the work of Preble and Casey as described in "Taking Care of Business—The Heroin User's Life on the Street" (1969), Cohen's *Delinquent Boys* (1955), and Robins' *Deviant Children Grown Up* (1966). Two of the most important emerging themes from this research offer a challenge to conventional thinking about the drugs-crime relationship:

- There is no single drugs-crime relationship. Rather, there are drugs-crime relationships, most of which are complex rather than simple.
- There is no simple solution to the complex challenges faced when drugs-crime relationships come into play.

By way of illustration, Brownstein and Goldstein offered and refined a tripartite conceptualization of drugs-crime relationships, which serves as a useful guide to some of the surrounding issues. Within this tripartite framework, one set of criminal offenses is described as psychopharmacologically induced (e.g., responses to intoxication states after drug taking). A second set of offenses is described as economic-compulsive in nature (e.g., instrumental income-producing criminal acts as needed to stave off symptoms of withdrawal states that appear once drug use has stopped). A third set of offenses is described as "systemic" and might be understood best as a consequence of a drug user entering or living within a social context in which extraordinary drug use is just one of a set of often intercorrelated criminal behaviors. That is, we do not need an appeal to drug intoxication, drug withdrawal states, or drug-induced compulsive behavior to account for offenses observed in this third category (Goldstein, 1985; Brownstein and Goldstein, 1990).

The tripartite framework clarifies three separate types of drugs-crime relationships, none of which is simple. As for analysis of simple solutions for these complex problems, a therapeutically oriented drug maintenance program might reduce the economic-compulsive type of offending without influencing the occurrence of crimes determined by poor judgment or other manifestations of intoxication states. A successful supply-side drug eradication program might reduce both pharmacological and economic-compulsive types of offending, but not offending of the systemic variety. Imprisonment of the drug user within a drug-free prison environment might extinguish today's crimes but might not influence tomorrow's offending when the prisoner is released back to the home community. Even if the prisoner remains drug free during the immediate postrelease period, the history of incarceration and a criminal record might constrain job opportunities and economic success to the point of inducing crimes that otherwise would not have been committed if the drug user never had been incarcerated in the first place.

Illuminated in this manner, the facets of multiple drugs-crime relationships become more clear; new opportunities for research open up. As these opportunities have been recognized, there has been a tremendous growth in scholarship and research activity on the topic of drugs and crime (see exhibit 1).

Scholars may benefit from an assembled listing or bibliography of this literature, now available in electronic form as a technical report from the Electronic Collaboratory for Investigations about Drugs at Johns Hopkins University (Forman, 2001). Readers interested in a recent comprehensive review of these publications can turn to the Harrison and Backenheimer-edited issue of *Substance Use & Misuse* on the drugs-crime nexus in the United States, which was published by Stanley Einstein and Marcel Decker, Inc., in 1998.

### **A conceptual framework for drugs-crime research**

Confronting the accumulated body of evidence and new literature, we have attempted to sort each element of evidence in relation to a conceptual framework originally devised for the field of psychiatric epidemiology and epidemiological research in general (Anthony and Van Etten, 1998). This conceptual framework is used as we train public health scientists for advanced research on drug dependence and related conditions. The framework may prove to be useful in the domain of criminal justice research as well, perhaps with suitable amendments by interested teachers and scholars.

**The rubrics.** Early in their public health research training, we ask our predoctoral and postdoctoral fellows to master the epidemiology of drug dependence. Here, drug dependence is defined as a syndrome or “running together” of clinical features, and sometimes is called drug addiction, especially when the focus is on such clinical features as obsession-like cravings and compulsion-like repetitive behaviors in which drug taking is central. The clinical features of the drug dependence syndrome include pharmacological tolerance, characteristic withdrawal signs and symptoms, almost obsessional thinking about drugs and drug-related behavior, and other observable mental, behavioral, and social adaptational manifestations of neuroadaptational processes that get started and progress with repeated drug taking.

This epidemiology of drug dependence is a subject matter to be mastered by the public health research fellows, just as they master the concepts, principles, and techniques used as methodological tools in the public health sciences. Mastery of this subject matter begins with study of the just-mentioned clinical features, the history of diagnostic criteria or case definitions used in public health research on drug dependence, and what has been learned about its neuroadaptational and genetic substrates. In the process, research fellows learn of patterned variation in drug dependence syndromes, some of which can be understood in relation to the pharmacology and pharmacokinetics of different drugs, such as cocaine versus heroin or methamphetamine versus oxycodone. Research fellows also learn about different measurement techniques used in laboratory, clinical, and field studies of the drug dependence syndromes. For example, under certain conditions, an appropriate dose of a narcotic antagonist can be used as a bioassay to check for the

presence of dependence on heroin or other opioid drugs (e.g., via precipitated withdrawal). Nonetheless, in general, the measurements of drug dependence rely heavily on self-report information obtained under specially protected confidential circumstances. To the extent that subjectively felt experiences such as “craving” and obsessional thinking about drugs are central clinical features for drug dependence, we cannot substitute human urine, saliva, or sweat samples for self-reports (Anthony, Neumark, and Van Etten, 2000).

Once issues of definition and measurement have been mastered, research fellows move on to what we call the “rubrics” of epidemiology—its main subheadings and associated research questions. These main rubrics and primary associated research questions are displayed in exhibit 2.

Successful research fellows learn these rubrics and use them to master not only the state of currently available evidence on each form of the drug dependence syndrome, but also the current gaps in evidence and the research concepts and tools needed to fill the gaps in evidence.

The relationship of each rubric to an associated set of research concepts and tools sometimes helps to clarify and differentiate the rubrics. Links between each rubric and corresponding research concepts and tools are presented in exhibit 3.

*Quantity.* Under the rubric of quantity, the main associated research question is “How many in the population are becoming affected, have become affected, and are now affected?” In this context, “becoming affected” can refer to becoming a drug user, developing drug dependence, initiating criminal behavior, or some combination thereof (e.g., see Gfroerer and Brodsky, 1992; Kosterman et al., 2000; Golub and Johnson, 2001a).

As reflected in the published scientific literature and technical reports made available by NIJ, the Substance Abuse and Mental Health Services Administration, and other Federal agencies, a substantial fraction of the Nation’s research expenditures on drugs and crime is directed toward the “report card” function of public health and criminal justice research under the rubric of quantity. A recent National Research Council report (Manski et al., 2001) tallied more than 60 Federal agencies with data systems designed to keep track of estimates on the number of drug users in households, among school-attending youths, among arrestees, among patients seen in emergency rooms, and in various other segments of American life. On the U.S. Department of Health and Human Services (HHS) side, we have the National Household Survey on Drug Abuse (NHSDA) with growing national probability samples of adolescents and adults (now with a sample size of more than 70,000 subjects per year); the Monitoring the Future (MTF) study, which started as a way to track drug use among graduating high school seniors through a national probability sample each year and now encompasses 8th and 10th graders; and a less intensive but more massive Centers for Disease Control and Prevention (CDC) surveillance of drug use and other health risk behaviors of teenagers in school. On the U.S. Department of Justice side, we have other ambitious survey operations, such as the Arrestee Drug Abuse

Monitoring (ADAM) program (formerly Drug Use Forecasting [DUF]), which monitors drug taking among arrestees through both self-reports and bioassay techniques, and the National Crime Victimization Survey.

Evaluated as part of the public health and criminal justice research enterprise, these substantial efforts may be understood best as examples of surveillance operations. The label “surveillance” does not trivialize the important work of the professionals and scientists whose daily labors, year in and year out, yield the hard-won surveillance data. In fact, many of our country’s surveillance operations in this domain of inquiry truly are gems and tend to be regarded as the best of the best in the world. In some respects, they are the envy of the world. Nonetheless, by definition, surveillance activities are designed with timeliness and practicality in mind, sometimes with deliberate decisions to sacrifice validity of measurement in favor of enhanced survey response rates.

For example, NHSDA, MTF, and the CDC survey all use self-report methods to measure drug-taking and crime-related behavior (e.g., weapon carrying). The option of bioassays to confirm self-report data now is regarded as impractical or too costly for surveys on a mass population scale, and there has been concern expressed that bioassays might reduce survey participation rates below acceptable values. NIDA is engaging in survey research experimentation with bioassays to complement self-report data to assess practical questions of this type. In the meantime, serious concerns have been expressed about the capacities of these data systems to provide evidence for policy evaluation (see, e.g., Manski et al., 2001). Nevertheless, evaluated from the standpoint of original plans for the data, these criticisms are somewhat impertinent as surveillance indicators. The criticisms are asking the surveillance operations to do far more than they originally were designed to offer.

The first rubric of epidemiology also encompasses studies of birth cohorts that are intended to estimate risks of adversity, plot trajectories of normative development, or quantify important population characteristics such as rates of officially recognized offending. The concept of a cohort study is familiar to criminal justice researchers and public health scientists alike. Prominent examples in the criminal justice research arena include Robins’ classic nonconcurrent cohort study of children seen by child guidance workers in the early 20th century (1966), and the work of Tracy, Wolfgang, and Figlio entitled *Delinquency Careers in Two Birth Cohorts* (1990).

The fact that the rubric of quantity is mentioned first does not mean that research under this rubric is easy or a methodological snap. Not at all. From the standpoint of data gathering, those of us who have recruited, trained, and supervised teams of 60 or more field worker-interviewers and quality control staff for data entry, documentation, and management can appreciate the operational challenges in surveillance work. From a statistical vantage point, the nature of the surveillance operations often includes interdependent observations within samples (e.g., sampled students within samples of schools; sampled household residents within neighborhoods; multiple respondents within sampled households, emergency rooms, or criminal justice facilities). These interdependencies motivate solutions that call on the calculus (e.g., in Taylor series

linearization for variance estimation). In some estimation applications, there is a need for Bayesian statistics not yet taught widely in graduate research training programs.

As to the importance of these “counting” operations, we may turn to a recent research contribution by Cohen, who incorporated values from these surveillance operations in his attempt to estimate the monetary value of rescuing a high-risk youth from a life of delinquency, crime, and other socially maladaptive behavior (1998). To complete this work, Cohen had to turn to an array of previous results from counting operations that ranged from the National Institute of Mental Health Epidemiologic Catchment Area surveys we conducted in Baltimore during the early 1980s with colleagues at four other university-based sites to work that Blumstein and his group completed to estimate basic parameters of criminal justice research, e.g., an estimated six percent of all boys account for more than half of all arrests (Blumstein et al., 1986).

There can be little doubt that investigators in the drugs-crime arena should be interested in Cohen’s conclusions about varying programmatic investments and the monetary returns from programs to intervene with high-risk youths. Nevertheless, it is somewhat startling to know that Cohen had to turn back to counting evidence gathered in the early 1980s and before to produce estimates to be used for policy and programmatic decisions almost two decades later. These quantitative estimates are not Avogadro’s number; rather, they are values expected to change over time, if not from place to place. If we value probing quantitative criminological research exemplified by Cohen’s work, then we must ensure that the drugs-crime research agenda includes periodic repetition of surveys to yield the required estimates.

Studying the accumulated evidence on the drugs-crime relationship, we have been able to sort much of it into the rubric of quantity. Quite clearly, NIJ and NIDA now make a considerable investment in the basic counting tasks required to estimate and quantify such parameters as how many adult arrestees and juvenile offenders are taking drugs each year. Each repetition of these surveillance operations provides evidence on variation in the estimates from time to time and from place to place. The study of this type of variation falls under the second rubric, which is called location.

*Location.* Our second rubric is location, and the main associated research question is “Where in the population are affected individuals more or less likely to be found, with variation in occurrence and frequency differentiated by characteristics of time, place, and person?” On occasion, work under this rubric is guided by theory, but more often the research has a more descriptive character.

James et al. (2002) provide an illustration of the nature of research and evidence about location. The research team set out to plot geographic variation in the occurrence of drug purchase opportunities experienced by young adults in the United States. In this figure, a “drug purchase opportunity,” a special form of drug-related crime opportunity, is measured by a survey response to a standardized assessment in interviews conducted for NHSDA. As depicted in exhibit 4, and substantiated with a univariate response regression model for description, there is variation in the occurrence of these drug purchase

opportunities across locational regions of the country and for young men versus young women. In this context, the statistical methods are not intended to probe the causes of the observed variation from place to place, nor the observed male-female differences. Rather, the methods are used simply to help quantify the uncertainty in the survey-based estimates and substantiate the presence of variation from place to place and the male-female differences (James et al., 2002).

This illustration is useful because it reminds us that location refers not only to geographic variation but also to variation in relation to individual-level characteristics (e.g., sex, age, socioeconomic status, ethnicity). For example, Fendrich et al. (1995) studied juvenile and older murderers to understand varying degrees of drug involvement in murder. Locational research also plots temporal changes, as illustrated in a recent NIJ report on the possibility of new marijuana epidemics, to be described below (Golub and Johnson, 2001b).

Estimates of the consistency of association between drug use and various arrest and criminal behavior types also serve to illustrate analyses focused on location within population experience: Crime was found to be more common among drug users than among nondrug users. As Harrison and Gfroerer (1992) make clear in their NHSDA analyses on this topic, the research questions they were trying to answer concerned the number of drug users, the number of individuals engaged in criminal behavior, and the overlap in these numbers. With respect to location, their work clarified the proportion of drug users who were engaged in criminal behavior and the prevalence of criminal behavior in relation to drug use. As is true in the work of James et al., these investigators did not draw on the apparatus of causal inference, matching, or other scientific maneuvers to disentangle whether the criminal behavior was a response to the drug use or vice versa. Nonetheless, taking a step beyond studies of officially recognized crimes, arrestees, and convicted criminals, Harrison and Gfroerer helped confirm links between drugs and criminal behavior, but they did not seek to produce definitive evidence about the causes of drug use or criminal behavior.

Much of our current research enterprise at the interface of drugs and crime has this type of descriptive character. Substantial HHS investments in the MTF study and NHSDA already have been mentioned. On the NIJ side, we call on ADAM to help clarify variation in the occurrence of drug use among arrestees across multiple jurisdictions, not only in the United States but also overseas. For the most part, we do not require these investments to yield definitive evidence that might be central in causal inference. Nonetheless, the evidence from these studies helps to describe the location of drug taking, criminal behavior, and the intersections of these behaviors, and sometimes to describe or predict the co-occurring and separate patterns of drug use and criminal behavior.

Analyses conducted under this rubric without a special push toward causal inference can be especially important in identifying hot spots within geocoded areas as well as health disparities that might differentially fall on one or another racial or ethnic minority group. Here, it is a predictive as well as a descriptive purpose that can be achieved. However,

when the task is to predict and not to explain, there is no special calling for the methods required for firm causal inferences, as depicted in exhibit 3.

Within the drugs-crime arena, there are many different examples of surveillance operations under the rubric of location, such as we can see in recent work by Golub and Johnson (2001b) in which they used DUF/ADAM data as evidence to advance their claims about a new and possibly expanding epidemic of marijuana use in the United States. True to the descriptive character of locational research, Golub and Johnson present evidence of the new and possibly expanding epidemic among offenders in some areas (e.g., Atlanta) and evidence of no epidemic in other areas (e.g., Miami), but they do not seek to explain why there should be an epidemic in one place but not in another. Because these data are from incarcerated individuals, an important set of complications arises in their interpretation. One suspects that the observed time trends and variation from place to place might reflect operations of local police departments as much or more than it reflects any underlying change in the dynamics of marijuana epidemiology.

This rubric of location also encompasses studies in which the investigators may be striving toward causal explanation, but they fall short, often demonstrated in a shift toward the language of “prediction” and away from the language of “explanation.” Two different hypothetical concluding statements can illustrate this point. When the research team falls short of its goal, the researchers may summarize their work by saying something like “Based on this study’s evidence, the level of drug use in early adolescence predicted later delinquency and criminal behavior in the young adult years.” A different verb is selected for the alternative, stronger form of concluding statement: “Based on this study’s evidence, the levels of delinquency and criminal behavior in the young adult years depend on levels of drug use in early adolescence.”

As the focus shifts from description or prediction toward explanation and causal inference, we move from the rubric of location to the third rubric of causes. The shift in focus calls into play a new set of research concepts, principles, and tools, as outlined in exhibit 3.

Many scholars will appreciate that a single study may contribute evidence under several rubrics at once. For example, the periodic reports of NHSDA, MTF, and ADAM routinely present evidence that falls under the rubric of quantity as well as the rubric of location. Rarely, the authors of these reports seek to make causal inferences from their surveillance data.

The yield of a study often is not clear at the outset or in the stages of study planning, and the study orientation to theory is not always a discriminating feature. Some theory-based studies have started as investigations of causes but have ended up making contributions solely in the domains of prediction and description. Other atheoretic studies end up making useful contributions in our studies of cause. Consider the first conclusive study on the topic of age-related risk of Down syndrome (DS) and associated mental retardation, completed some 50 years ago. The investigators sought to plot the risk of DS by the age of the mother at the time of delivery. An exponential increase in risk after age

40 was clear in the first graphs. We still do not know what causes the chromosomal trisomies that give rise to DS, nor do we know why these trisomies and DS are more common when older mothers give birth. But even in the absence of firm causal theory and evidence, it has been possible to reduce the occurrence of DS in human populations by encouraging mothers to bear their children before age 40. Hence, a strictly descriptive study provoked an effective intervention to reduce the occurrence of an important genetic condition.

It is regrettable that our studies of disparities affecting racially and ethnically defined subgroups of the American population generally fall under the rubric of location, as do our studies of the changing dynamics of household and family composition in the United States. For example, we now can say with some certainty that African-American males experience rates of arrest, prosecution, and incarceration for drug possession offenses that cannot be explained by their rates of drug taking, but we do not have good evidence on the causes of this racial disparity. Initial inquiries suggest differential law enforcement and judicial practices, which sometimes encompass racial profiling, but rigorous scientific evidence on these practices is scarce.

With respect to the dynamics of household and family composition, the phenomena of youthful drug taking and related criminal offending have links back to the families of origin, now often characterized by absence or infrequent appearance of the father in many of our population groups. This is not to say that female-headed households are homogeneous or uniformly deleterious with respect to socially maladaptive behavior of young people. It would be a mistake to presume that the traditional mother-father household always and in all contexts is more protective than a female-headed household with respect to the risk of youthful drug taking or delinquent behavior (e.g., see Chilcoat, 1992). Mothers often mobilize family resources or draw on assets that in some measure may help compensate for absent fathers (e.g., by involving grandparents, neighbors, church groups), as described by Kellam, Ensminger, and Turner (1977), Pearson et al. (1990), and others.

A research agenda on race, ethnicity, and family or household composition can be motivated by an awareness that the drugs-crime relationships will depend to some extent on demographic trends. Against the backdrop of demographic trends such as these, including an increased prominence of Hispanic children and families in the United States, it will be important to sustain the research agenda in the domain of locational variations of this type. Important steps in this direction have been taken in the Federal agencies responsible for surveillance of drug-related behaviors, including increased attention to measurement of ethnic self-identification (e.g., with respect to Cuban origin, Puerto Rico origin, and other subgroups of the Hispanic population; with respect to Chinese origin, Samoan origin, and other subgroups within the Asian-Pacific Islander category). Similar attention is required in criminal justice research such as ADAM and I-ADAM (International ADAM) and in administrative statistics compiled on operations of the criminal justice system in this country.

Whereas the human genome project is challenging conventional views about “race” as a scientific concept, studies on self-identified race-ethnicity will have a sustained importance in the NIH-NIDA research agenda on the topic of drugs and crime. This evaluation of importance can be grounded in an awareness of the demographic trends described above, but it also draws on an appreciation of what studies of self-identified race-ethnicity may teach us about the influence of cultural contexts and socially learned behaviors with respect to drug taking and criminal behavior.

Finally, a note on ethnographic studies should be added here. In general, the sample size and “sample space” characteristics of these studies do not make ethnography an especially fertile discipline with respect to the first rubric of quantity, except when the characteristic under study has extremely limited dispersion. For anyone who looks to ethnographic studies for quantitative values, there often are some unanswerable questions about generalizability and precision of the study estimates. In some respects, ethnography might be characterized as a search for the boundaries of no variation in a socially shared human characteristic.

This is not to say that ethnography is barren when it comes to quantitative data. To the contrary, the small scale of ethnographic research makes it possible for ethnographers to shift directions more quickly than is possible in ordinary surveillance operations. As a result, ethnographic field workers helped in the early identification of crack cocaine, methamphetamine, and oxycodone outbreaks—years before these outbreaks could be identified in large-sample surveillance data.

Under the rubric of location, ethnographic field workers were among the first to note inner-city adolescents whose drug taking started with marijuana rather than with the more normative experiences with alcohol and tobacco. They also were the first to characterize a growing use of “blunts”—tobacco cigars hollowed out and filled with marijuana for a combined tobacco-marijuana intoxication (Golub and Johnson, 1999). In a recent round of observations, there is a suggestion that for some youths, the typical “gateway” drugs have been skipped—an example of subgroup variation in the more typical developmental sequences running through alcohol, tobacco, and marijuana to drugs such as heroin, stimulants, and hallucinogens. Large-sample epidemiological surveillance data now seem to confirm the initial ethnographic observations on this topic (e.g., see Golub and Johnson, 1999; Golub and Johnson, 2001a).

One of the reasons ethnographic research is important under the rubric of location is that it can open our eyes to new conceptions of time, place, and personal characteristics that impinge on the drugs-crime relationship. These ethnographic studies are especially useful in descriptions of the cultural context and socially learned behaviors described above. Their evidence can add depth and insight to otherwise superficially understood intersections of drug taking and criminal behavior.

*Causes.* The third rubric of epidemiology pertains to the study of causes and draws on the research apparatus required for causal inference (exhibit 2). On occasion, this research apparatus can be quite simple in concept. For example, a relatively small sample of

monozygotic (MZ) twins discordant for an important outcome is sufficient to provide definitive evidence about environment with respect to the causes of that outcome. These MZ twins are genetically matched: If they are discordant for outcome, one may look for gene-environment interactions, but more often one looks for differences in environmental conditions in utero (e.g., dichorionic versus monochorionic sacs), perinatally (e.g., insults at the time of delivery), or in later development (e.g., head trauma for one twin but not the other during infancy or childhood). The National Institutes of Health (NIH) investment in recent twin research to estimate heritability of different forms of drug use now generally is paying off in two ways:

- Each study is indicating at least some degree of heritability of drug dependence, and sometimes heritability of drug use, especially legal drug use (e.g., tobacco).
- Each study is indicating ample room for gene-environment interaction or for influence of environmental conditions and processes.

These results from causal research help substantiate a case for a future research agenda on the genetic sources of variation and on environmental modulation of these genetic sources of variation.

Randomized trials with relatively simple structure also can be used to probe causal hypotheses with definitive results. For example, these trials may offer our best avenues toward definitive evidence on whether cessation of illegal drug use is followed by reductions or elimination in criminal behavior. An alternative is to nest the study of causes within a more expanded agenda of systems research on drugs-crime relationships (Manski et al., 2001). To the extent that systems research entails a finely detailed specification of mechanisms that link events and processes within a system, this type of research falls more clearly under the rubric of mechanisms, as described below.

Outside of the simplicity of research on discordant MZ twins and randomized controlled trials, a complex apparatus of study design and statistical method is required to extract definitive evidence in research on drugs-crime relationships. Given the importance of inferences about causes in the drugs-crime relationship, it may be understandable that graduate research training programs have become increasingly methodological in their orientations.

It may be appropriate to discuss the potential contribution of ethnographic research in relation to the causes of the drugs-crime relationship. To date, most ethnographic research on drugs and crime has been descriptive in character. It has provided leads for more probing causal investigations, but it has not produced definitive evidence on the links between drugs and crime. In this respect, ethnography's contribution may be most important under the rubric of location. Before anyone could mobilize large-sample surveillance operations to study the new drugs-crime phenomena connected with crack cocaine (e.g., crack and prostitution), it was possible for ethnographers to move in and make headway. To some extent, ethnographers have been pioneers in research on methamphetamine and club drugs such as MDMA (Ecstasy), and we can expect more of

the same in relation to our first new drugs-crime outbreaks of the 21st century, which involve sustained release oxycodone.

An NIH-NIDA investment in ethnographic research on drugs-crime relationships of this type will continue to be important—if only to help us begin to understand the unusually circumscribed geographic distributions of methamphetamine and oxycodone use in the United States and the patterns of criminal behavior associated with use of these drugs. Ethnography can be used to produce a catalog of causal explanations for methamphetamine's emergence as a threat to public health and public safety in rural sectors of the American Midwest and for oxycodone's emergence in small cities and towns of the Appalachian mountain range, especially from West Virginia southward. It is not clear that ethnography or any other scientific field will be capable of producing definitive evidence about specific explanations in this catalog of causes. Nonetheless, there is value and importance in the attempt to do so, and ethnographers can bring rigor and scientific discipline to this process of investigating these causes. The alternative seems to be to leave these investigations to the field of journalism.

*Mechanisms.* Within epidemiology generally, mechanisms refer to linkages of states and processes that lead toward expressions in clinical features of health and illness or disease. As applied to the drugs-crime relationship, one might ask about the mechanisms of linked states and processes leading to or away from an association between illegal drug use and criminal behavior.

For an illustration of these mechanisms, one may turn to the coercive process and deviancy training models introduced in the work of Patterson and Dishion. Their Oregon Boys study has provided longitudinal evidence on what surely must be central linkages in the mechanisms underlying drugs-crime relationships (e.g., see Patterson, Dishion, and Yoerger 2000). For example, studying these school-based samples of boys through ages 17–18, and using standardized coding of a 30-minute free discussion-interaction between best friends, they found substantial over-time correlation of deviant friendship process (e.g., duration of rule-breaking talk bouts as coded from videotape). Dishion also has reported on a link from initial drug use to increased affiliation with deviant peers and onward to initiation of criminal behavior that is more consistent with the delinquency-to-drugs link that emerged in the longitudinal research of Johnston and colleagues based on MTF analyses published more than 20 years ago (Dishion et al., 1996; Johnston, O'Malley, and Eveland, 1978), as well as on more recent studies (e.g., Elliott and Huizinga, 1989).

The use of multiwave longitudinal study designs to probe into suspected causal mechanisms is well known in both public health and criminal justice research circles. The Alcohol, Drug Abuse, and Mental Health Administration (the precursor to SAMHSA), and more recently NIH and OJJDP have maintained support for a series of important longitudinal studies over the years (e.g., see the work of Jessor and Jessor, Kellam and Ensminger, Block and Block, McCord, Bachman, Kandel, Robins, Elliott and Huizinga, Hawkins and Catalano, and many other studies of this type, as listed in compendia such as Verdonik and Sherrod, 1984). Advantages of long-term investment in these

longitudinal studies can be seen in the research articles from many of the research projects with multiwave assessments, for example, the Pittsburgh Youth Study (e.g., Loeber et al., 1998), the Denver Youth Study, and the Rochester Youth Development Study (Loeber et al., 1999); and the research groups led by the Brooks, Newcomb, and Bentler (e.g., see Brook et al., 1996, Brook et al., 2000; Newcomb and Bentler, 1988; Newcomb 1992).

One of the questions in the design of an agenda for future research on drugs and crime is how the evidence from large- and medium-sized samples from longitudinal studies of this type might be linked with evidence from the generally much smaller intensive studies of cases. Until there is consensus about effective interventions to disrupt the drugs-crime relationship, possibilities for a linkage exist through the concept of natural history.

In the history of medicine and medical research, the first natural historians of disease were clinicians and clinically oriented observers who made careful observations at the bedside of patients, in the absence of effective interventions. They watched, measured as best they could (e.g., body temperature), and described change in relation to the passage of time from the first recognition of clinical features. Within the realm of drugs and crime research, ethnographers and social scientists generally have taken over the responsibilities of careful clinical observers in relation to illegal drug use and criminal behavior. During the last 50 years, thanks to the work of Robins (1966), Winick (1962), Preble and Casey (1969), Agar (1973), Waldorf (1998), Nurco (Nurco et al., 1975, 1996; Nurco, 1998), and their successors, we have learned much about the natural history of drug use, drug dependence, and associated criminal behavior through ethnographic and social science research.

The natural history of a disease proves to be an important element under the rubric of mechanisms. In the past, a careful description of a disease's natural history often has guided investigators toward underlying causal mechanisms.

In the years before effective drug treatments, Winick and others drew attention to the maturing out process for drug addicts, and there is a parallel literature on maturing out with respect to criminal behavior in general (Winick, 1963). The maturing out process continues to be an important locus for new research on the drugs-crime relationship.

Other clues about causal mechanisms are being produced in observational and longitudinal studies of individual cases or families characterized by some feature of the drugs-crime relationship. For example, we have Dunlap's intensive studies of families in which one of the members is a crack cocaine dealer (Dunlap and Johnson, 1996; Dunlap, Johnson, and Manwar, 1994); research such as Spunt's study of adolescent offenders with a history of violent crime (Spunt et al., 1990), Longshore's linkage of DUF and California Bureau of Criminal Statistics data (Longshore 2000), and the earlier related studies started by Hser, Anglin, and McGlothlin (1987); and investigations led by Inciardi, Johnson, and Goldstein or members of their research groups (e.g., see Inciardi and Russe, 1977; Inciardi 1990; Inciardi and Pottieger, 1998; Johnson, Dunlap, and Maher, 1998; Goldstein 1998; Spunt et al., 1990, 1994, 1995).

Several interesting elaborations of these intensive case studies have developed in the realm of criminal justice research. For example, Logan (2001) has added bioassays for metabolites of the neurotransmitter serotonin as well as testosterone assays as part of his intensive followup studies of crack users. This example serves to illustrate a potential intersection of public health and criminal justice research that should be explored in more depth as we work through a future agenda for research on drugs and crime.

A conceptual shuttling back and forth between these intensive smaller sample studies and the generally larger sample longitudinal studies would seem to have advantages for investigators who work in one or another of these arenas, and there are a few investigators who conduct both types of studies (e.g., see Dishion and Loeber, 1985; Dishion, Patterson, and Reid, 1988; Dishion et al., 1996). This type of bridgework between the microsocial and ethnographic research traditions and large-scale longitudinal sample research deserves to be a deliberate focal point on the future drugs-crime research agenda. This focal point is important because the study of causal mechanisms and processes can draw attention to potentially vulnerable links where new interventions might be directed.

In epidemiology generally, the focus of research on causal mechanisms is shifting to genes and encoded gene products, as displayed in our most recently emerging subspecialties of genetic epidemiology and molecular epidemiology. To some extent, Elliott has a head start in a potential cross-fertilization between criminal justice research, genetic epidemiology, and molecular epidemiology. He already has introduced harvesting of DNA samples in the context of his national longitudinal study (Elliott, 2001). Opportunities for case-control studies and other genetically informative designs, including whole genome scans nested in a case-cohort study design, will become possible as this research evolves. Eventually, this type of work should lead us toward more definitive evidence on causal mechanisms underlying the drugs-crime relationship, including gene-environment interactions.

It is possible to make a forecast of likely integrations of genetic research, cognitive sciences, and the more traditional disciplines of behavioral and social sciences for a future agenda for NIJ and NIDA research on drugs-crime relationships. For example, exhibits 5–7 represent an elaboration of conceptual models our research group has developed as an aid to our study of transitions from drug use to drug dependence. Exhibit 5 expresses a suspected causal influence of drug use on criminal behavior. It also expresses a separate influence of drug dependence on criminal behavior. These two specifications are consistent with the Goldstein-Brownstein distinctions between drug-related crimes that might arise from acute drug intoxication states versus crimes that are rooted in the economic-compulsive behavior of an individual who suffers withdrawal states as a result of sustained drug use and neuroadaptation. There are many law-abiding drug dependent individuals who do not commit crimes, even when they are suffering from withdrawal pains. Hence, exhibit 5 includes a speculative causal pathway that runs directly from withdrawal to the occurrence of criminal behavior, over and above the

separately specified role of the drug dependence syndrome for which withdrawal serves as a manifest indicator.

We speculate that an individual's genome can contribute to the drugs-crime relationship in different ways. Exhibit 5 concentrates on a possibility that some genetic polymorphisms or mutations may be intercorrelated manifestations of an underlying diathesis or vulnerability to make the transition into drug dependence from a state of nondependent drug taking, as reflected in pathway 1. It also specifies a possibility that a specific polymorphism (or mutation) has an additional influence on this transition, as reflected in pathway 2. As indicated by pathway 3, we may hope for development of effective intervention techniques that can disrupt what otherwise might be an expression of the diathesis. If effective, these interventions will slow or disrupt the natural history of drug dependence at a step in the process that links nondependent drug taking and the subsequent transition into drug dependence. This effect of intervention, by itself, may be sufficient to alter the drugs-crime relationships depicted to the right of the exhibit.

The potential role of the cognitive sciences is expressed in the intermediate pathways that link nondependent drug taking and drug dependence to later criminal behavior. Here, aggression may be conceptualized in a generic sense as rowdy misbehavior or social maladaptation secondary to drug taking, which can occur with or without criminal behavior. Executive dysfunction refers to impairments in the cognitive processes that subserve human capacity to plan, direct, and control one's future behavior within adaptational boundaries and may encompass more generalized planning behavior (e.g., see Tolman, Edleson, and Fendrich, 1996).

As depicted in exhibit 5, during states of acute drug intoxication, there may be a release of aggressive behavior and a disruption of regulatory executive functions. As levels of drug dependence increase, levels of aggressive behavior can change in an upward or downward direction and executive dysfunctions can occur. The complexity of interrelationships between aggression and executive dysfunction is reflected in the reciprocal causal paths between these two constructs. Increased executive dysfunction translates as inept decisionmaking about aggression and the subjective utility functions that govern decisions about whether to commit a crime. As part of generally adaptive fight-flight responses and modulation of monoamine neurotransmitter signaling pathways during bouts of aggression, there can be a cascade of executive dysfunctions: Mere rowdiness can be transformed into aggravated assault.

To be sure, exhibit 5 is only a model that represents little more than an oversimplified representation of the complexities that link an individual's genome with cognition and behavior. Models by definition are oversimplified representations. It is fair to ask whether the model requires additional specifications, such as the possibility that religious convictions might tend to modulate the relationship between drug taking and aggression or criminal behavior. In this oversimplification, exhibit 5 does not convey all such possibilities. These possibilities for elaboration of the longitudinal model should help the reader understand some of the complexities faced in observational studies of causal mechanisms that account for observed drugs-crime relationships.

Exhibit 6 presents even more simplification to sharpen focus on the drugs-crime relationship specifically. The genetically based diathesis and other covariates of exhibit 5 are set into the background (i.e., presumed to exist but not explicitly depicted). In exhibit 6, we see a readily appreciated reciprocity between the level of drug taking and the level of drug dependence: (a) the more drug taking, the more we find increased drug dependence levels, and (b) the more drug dependence, the more we find increased levels of drug taking. We also see the level of criminal behavior expressed as a function of levels of drug taking and drug dependence, as shown in exhibit 5. An additional elaboration involves the longitudinality of this model. We have levels of criminal behavior at one point in time influencing levels of criminal behavior at future points in time, but in exhibit 6, we do not specify a link from levels of criminal behavior to subsequent drug taking or drug dependence levels. At least in theory, and in some prior suggestive evidence, this omission represents a potentially important mis-specification of our model for the drugs-crime relationship (e.g., see Johnson et al., 1995).

Exhibit 7 adds a level of complexity to the model depicted in exhibit 6 and poses a substantive question for the agenda of action research: “How might an intervention lead to change in this system of interrelationships?” We introduce the possibility that social status (e.g., status in the community, socioeconomic status, lawful income) depends on criminal behavior and also on the level of drug dependence, and that criminal behavior influences the subsequent level of drug dependence by its intermediate influence on social status. The model depicted in this exhibit also provides for a plausible link from the level of drug dependence to subsequent criminal behavior. Namely, as drug dependence increases and social status (e.g., lawful income) falls short, criminal behavior may increase (as in the Goldstein-Brownstein tripartite model). In addition, subsequent levels of drug dependence may be influenced by the changes in social status, either upward or downward.

The model in exhibit 7 also introduces a conglomerate concept of “assortative peering,” expressing a well-known truism: “birds of a feather flock together.” The occurrence of drug taking is linked to later formation of peer group relationships, as is the occurrence of criminal behavior. To some extent, we can say that past drug use and past criminal behavior influence current peer group affiliations, and to some extent, we can say that past peer group affiliations influence future drug use and future criminal behavior. These complexities are expressed by hypothesized causal paths in exhibit 7.

Conceptual models of this type are incomplete representations of the causal mechanisms that lie beneath observed drugs-crime relationships, yet they are elaborations of the Goldstein-Brownstein tripartite model. Nonetheless, most readers will agree that these representations are oversimplified. If they have value, it is to highlight some future directions for the joint NIH-NIDA research agenda on drugs-crime relationships.

We do not yet have a longitudinal research program to investigate the relatively simple model of interrelationships between levels of drug use, drug dependence, and criminal behavior as depicted in exhibit 6, let alone the more complex model of exhibit 7, with its

sociological construct of social status and the social psychological construct of affiliation with behaviorally similar peers (assortative peering, homophily, etc.). Fortunately, there already is a cadre of criminologists and drug researchers who are trained in sociology and social psychology and can readily incorporate the biomedical and clinical concepts of drug dependence into their research plans, if supported to do so.

It will be more difficult to forge a research agenda that integrates the genetics research and cognitive sciences constructs depicted in exhibit 5. For the most part, genetics and cognitive sciences are unknown territories for most NIH and NIDA investigators who have made important contributions in past research on the drugs-crime relationships. For most drugs-crime researchers, it would not be difficult to integrate concepts and measurements of aggressive behavior and the clinical syndrome of drug dependence within their existing research plans. Far more difficulty will be encountered during the process of integrating genetics and the neuropsychological and neurophysiological measurements of the cognitive sciences.

We can learn a lot about the drugs-crime relationship simply by replicating and refining important longitudinal research on drugs-crime relationships that was initiated during the second half of the 20th century. Many of these longitudinal studies have cohorts that still are intact, and followup studies are now underway to learn more as these cohorts mature into adolescence and make the transitions into young and middle adulthood. There are mountains of data from 20th-century studies that have not yet been fully exploited through careful analysis.

Nonetheless, as we look forward through the next decades of research, the NIH-NIDA agenda must go beyond what has developed as the best 20th-century research on the drugs-crime relationship. Ten decades from now, if we are to leave the 21st century with an enhanced understanding of the drugs-crime relationship and with a greater capacity for effective action to improve public health and safety in this domain, we cannot continue to work within the narrow paradigms and methodologies of the traditional scientific disciplines mastered by drugs-crime investigators of the 20th century. If we are successful, then in a few decades, the biomedical, genetic, and cognitive science substrates of the drugs-crime relationship will no longer be a matter of mere speculation, as depicted in exhibits 5 and 7. There will be definitive evidence, solid understanding, and effective action-plans based on what we learn from the pioneers who move into that now-unexplored territory.

*Prevention and control.* The long-term value of research on causal mechanisms depends on identifying potentially vulnerable linkages in the sequence of states and processes that lead to illegal drug use and criminal behavior. It may go without saying that increasingly definitive evidence about causes and causal mechanisms will help us achieve our goals in the domain of effective prevention and control. Nonetheless, a reminder may be useful with respect to a dynamic interrelationship between etiological studies (of causes) and the emergence of effective interventions. As illustrated in the circumstance of DS and maternal age, with limited evidence on the underlying causal mechanisms of DS, by manipulating maternal age we have a very effective instrument to prevent and reduce the

risk of DS. As explained in our original paper on the rubrics of epidemiology, many effective public health preventive interventions emerged before firm knowledge about causes and causal mechanisms became available (Anthony and Van Etten, 1998).

A related concept involves the use of randomized preventive trials to provide increasingly definitive evidence about suspected causal relationships. Some readers of this paper will know of work that Kellam and our Johns Hopkins research team have completed, using randomized field trials to probe the interrelationship between early aggressive and rule-breaking behavior and later drug involvement among boys (e.g., Kellam and Anthony, 1998). In essence, we decided that more observational research on the link from early aggression or deviance and later drug use would be less important than an experimental test. Within the framework of a randomized field trial, we tried to and succeeded in reducing aggressive and deviant behavior of first-graders using an experimental intervention assigned at random. For the boys assigned to experimental intervention, we have found later reduced occurrence of drug involvement, and we have replicated these results in a second cohort of first-graders (Kellam and Anthony, 1998). More replications along these lines are needed before anyone can claim that early aggression or deviance is a “cause” of later drug use, but this experimentation illustrates how experimental research in the domain of prevention and control can yield benefits in the form of improved evidence to test causal theories. This idea is not new. Hawkins, Catalano, Offord, and others have noted it as well (e.g., Hawkins, Von Cleve, and Catalano, 1991; Hawkins, Catalano, and Miller, 1992; Jones and Offord, 1989). But it is an idea that often is overlooked by investigators more interested in theory testing and who orient themselves toward goal 1, described in this paper’s first paragraph. Under the fifth rubric, we try to orient the research to serve both goal 1 and goal 2.

Because elements of this rubric of prevention and control are being covered in the companion papers that accompany this working manuscript, I will close this section more quickly than might be customary. Before doing so, I would like to mention the contributions of operations research and systems research in this domain, which often have been neglected in epidemiology. Over the years, the thoughtful and quantitatively sharp work of Blumstein and colleagues has continued to inspire an important line of research on prevention and control that is pertinent to the drugs-crime relationship. Although I am not confident about all of the data or assumptions of the underlying analysis approaches, I have been especially impressed by the directions taken by Blumstein colleagues Caulkins and Cohen in this regard.

For example, Cohen (1998) discusses potential synergy of programs and distinguishes the aggregate benefits of programs designed to reduce crime versus the sum of the benefits of individual programs. It is possible that no single program would help city residents feel safe enough to derive lifestyle-related expenditure benefits (e.g., walking a mile through a rough neighborhood versus taking a taxicab because of concerns about safety). Combinations of programs might do so. This distinction ties into the concept of marginal costs versus average costs associated with drug-using and delinquent youths or criminals, where the marginal costs exclude fear of crime and private security expenditures because these costs are largely unaffected by any one criminal’s actions.

Caulkins and his colleagues developed a challenging line of systems research that can ultimately yield new ideas and evidence about policy instruments in relation to the drugs-crime relationship. The evolution of this work toward selection of policies and programmatic instruments at different stages of a drug-taking epidemic is especially important (Caulkins, Crawford, and Reuter, 1993; Behrens et al., 1999).

## **A Selective Overview of Tensions Faced in Research on Drugs and Crime**

Numerous tensions are faced at the intersection of public health and criminal justice research on the drugs-crime relationship. This section identifies and describes a selection of these tensions, and in some instances recommendations are offered for NIJ and NIDA action to help resolve the tensions.

### **Tensions in theoretical perspectives, concept, and definition**

Heterogeneity at the intersections of public health and criminal justice research is not limited to differences of opinions and judgment about empirical observations, the inferences we can draw from these observations, and the uses to which we apply the observations (e.g., cost analyses of alternative programs). There are some fundamental tensions within and across theoretical perspectives and also approach.

**The concept of scale.** Ecologists work with a concept of scale that may help us understand some of the tensions mentioned above and may serve as an axis of orientation as we turn to future directions for research (e.g., see Brown, 1995; Wiens et al., 1986). As a concept, scale resonates with what educational researchers and behavioral and social scientists often have termed multilevel or hierarchical models, as in Ennett's and the Duncans' research with young people nested within ecological niches of higher order such as classrooms, schools, or families (e.g., see Ennett et al., 1997; Duncan et al., 1997; Duncan, Duncan, and Hops, 1998) and our own research group's nesting of individual drug users and collections of drug users in their neighborhoods of residence (e.g., Bobashev and Anthony, 1998; Petronis and Anthony, 2000). Parker and Toth (1990) also have appealed to related macro versus micro concepts in their research on alcohol and homicide, as have Patterson and colleagues in their research on peer groups (Patterson, Dishion, and Yoerger, 2000). Bronfenbrenner's ecological systems theory for human developmental research slices scale into macro, meso, and micro divisions that many investigators have found useful (Bronfenbrenner, 1979, 1986).

Although not with any direct reference to a formal ecological concept of scale, we can see resonance of this concept in Markowitz and Grossman's studies of taxes and regulations on alcohol and their hypothesized effects on criminal behavior (Markowitz and Grossman, 2000), the research of Caulkins and colleagues on national drug policy and programmatic initiatives (e.g., Behrens et al., 2001), and Holder's research on preventive interventions directed toward communities in the United States (Holder 1993, 2001; Holder et al., 1999, 2000). Scale is worked outward from the individual organism in the direction of larger social groups, organizations, and geopolitical units. In public health and criminal justice research, we often refer to pre-established institutional or

geopolitical boundaries (schools, census tracts, nations) when we work at higher scale. In ecology, mathematical models and methods such as advanced wavelet analysis are used to allow the empirical data to inform scale—as in research on landscape ecology (Anthony and Bradshaw, 2001).

Some tensions arise in research when investigators ignore scale in their theoretical perspectives or empirical research reports. For example, most of us work within a conceptual framework that leads us to comprehend estimates of the drugs-crime relationship without reference to scale. However, one should expect the drugs-crime relationship to be of one order of magnitude when we are investigating individuals who all reside in the same local area (e.g., as in much of the ethnographic research on the drugs-crime nexus), a different order of magnitude when we work with individuals and data from multiple neighborhoods, but with matching on neighborhood in the analysis, and a different order of magnitude when our data are from individuals across the Nation, with no analytical attention to who lives near whom, except perhaps during the process of estimating variances for confidence limits and standard errors (e.g., see Bobashev and Anthony, 1998).

Although not clearly within the scope of the original ecological concept of scale, an investigator may work inward from the boundaries of the whole organism toward subunits, ultimately leading to the signaling pathways between neurons, messenger systems originating from genetic material, and the simplest proteins and the encoding genes themselves. This elaboration of the concept of scale creates yet another tension, in part because the concepts of genetics and signaling pathways for neurotransmission are more familiar in the public health research community but are not yet in the mainstream of graduate or postdoctoral research training in the criminal justice research community. To illustrate, when I have talked with my criminal justice research colleagues about Elliott's inclusion of DNA assays in the most recent waves of his National Youth Survey, many of them have asked, "Why?" To be sure, some skeptical behavioral genetics colleagues also have asked, "Why?" but this is an instance in which the same verbal behavior has origins in substantially different theoretical models. My point is that tension can arise when concepts of scale are not made explicit.

Some of the work at the intersections of public health and criminal justice research will be to make our concepts of scale explicit. In some respects, this will be more readily accomplished as we work from the whole organism outward, and the task may be more difficult as we try to integrate molecular biology, genetics, and neuroscience into our discussions. Nonetheless, this hard work will be essential as we make a 21st century science of drugs-crime relationships.

**Orienting definitions and constructs.** The literature also displays considerable heterogeneity in orienting definitions and constructs. On the public health side, there often has been an orientation toward drug use and drug dependence or addiction as useful constructs in their own right. One orientation often has been called the "medical model," but it amounts to little more than an analysis of empirical syndromes (i.e., co-occurring manifestations of the neuroadaptational processes that get started when drug use begins,

followed by a cascade of secondary and tertiary adaptations, some of them occurring in the domain of social adaptational roles and responsibilities). In a later section of this paper, I will return to this syndrome concept. On the criminal justice side, drug use and the drug problems associated with drug use often are treated as if they are not interesting in their own right but are something akin to interchangeable observable manifestations of something else that is more fundamental, such as the “problem behavior syndrome” construct first elucidated by Jessor and Jessor (1977) some 30 years ago. A more recent version of this concept is a general deviance construct used by Scheier, Botvin, and others in empirical studies (e.g., see Scheier and Botvin, 1996), and there also is a recent respecification of the Jessor and Jessor model, with elaborations that encompass the epidemiological concepts of risk factors and protective factors (Jessor, 1998).

The literature also shows heterogeneity in the typologies of criminal behavior or social maladaptation. Notions of childhood conduct disorder followed by Antisocial Personality Disorder appear prominently in some formulations, but are absent elsewhere (Loeber and Schmalings, 1985; Stevens, Kaplan, and Bauer, 2001; Langbehn and Cadoret, 2001).

These definitions and constructs in our theoretical perspectives demand work at the intersection of public health and criminal justice research. If we cannot bridge these different approaches or marry them to produce adaptive offspring, they will prove to be an unending source of unresolved tension with implications for research progress. Unresolved tensions slow down our progress in research that depends on a peer review process, whether the peer review occurs at the stage of reviewing proposals or of vetting journal articles.

At NIJ and NIDA, an important part of the research agenda can be a series of meetings or technical workshops. The charge to workshop participants is to bridge these orienting concepts and definitions across disciplines or create an articulation between concepts that will accelerate research progress on drugs-crime relationships rather than slow it down.

**Orienting conceptual frameworks and theories.** The originating biomedical branches of public health research sometimes take theory as a given or work with theory in the background when there are emergent problems of human suffering and disease to be solved. For example, the important 20<sup>th</sup>-century line of investigations required to identify lung cancer as an adverse consequence of tobacco smoking was guided more by implicit concepts of carcinogenesis secondary to tobacco smoking. Strongly articulated, explicit theories, if any, would have been mis-specified and incomplete in that they could not possibly have incorporated the postsmoking DNA adducts, protein adducts, and gene-encoded metabolizing enzymes now prominent in the models of carcinogenesis. In criminal justice research, true to its origins in the social and behavioral sciences, the theoretical underpinnings are made more explicit (e.g., see Thornberry, 1997; Kaplan, 1995). One might say that without explicit theory, the research in this domain stands little chance in peer review, no matter how important the empirical contribution.

This is another source of tension at the intersections of and sometimes within the domains of public health and criminal justice research. In Public Health Service study sections, I

have seen study section members be less than enthusiastic about proposed epidemiological research on drug use and Antisocial Personality Disorder because the applicants had not oriented themselves to the major theories of deviance well known in criminal justice circles: “inadequate conceptual model” is the phrase that comes to mind. I also have observed major differences of opinion about scientific priority among experts in the criminal justice and social science world, some of whom are comfortable with “psychologizing” constructs within their theories (e.g., the self-derogation models developed by Kaplan), and others who are more focused on constructs with a behavior analytic origin (e.g., coercive process and deviancy training models developed by Patterson, Dishion, and their research groups in Oregon).

Tension in relation to theoretical models has been readily apparent in this NIJ-NIDA collaboration, which has offered a chance to step back and look over a broad expanse of scientific progress in public health and criminal justice research on the drugs-crime association. This broad perspective creates germs of ideas that might be useful in a synthesis or integration of various theoretical perspectives that range from the disciplines of molecular or behavioral genetics to those of econometrics and the other social sciences. However, there clearly is diversity and tension even within fields as narrow as behavior genetics, where some work is oriented toward developmental family processes (e.g., as advanced in the recent work of Neiderhiser and colleagues), and other work is not (e.g., see Neiderhiser et al., 1998, 1999; Neiderhiser, 2001; Brennan, Mednick, and Jacobsen, 1996; Tehrani et al., 1998; Kotler et al., 1999).

These tensions surface most clearly in debate and discussion of an intersection of genetics research and studies of the drugs-crime relationship. Many investigators from social science backgrounds are hesitant to take part in discussions of genetics, gene expression, and mechanisms of inheritance that might account for covariation of drug-taking behavior and criminal offending. This hesitation can be traced in part back to serious and important concerns about ethical issues, eugenics, and the like. Some of the hesitation can be traced back to a gap in graduate education: Graduates of social science training programs often have not mastered the basics of human biology and genetics.

Looking from a different perspective, an observer can see other sources of tension in relation to conceptual framework and theories. Graduates of human biology and genetics programs often have not mastered the basics of behavioral and social sciences research.

The intersection of the Human Genome Project, gene expression, and proteomics with research on drugs-crime relationships merits close attention at NIDA and NIJ. To some extent, this intersection can be cultivated in a gradual process of shaping new investigators. NIDA’s peer review of its portfolio of research training programs and individual career development awards can specify requirements for cross-discipline mastery. On one side, new social science investigators can be required to master the basics of human biology and genetics. On the other side, new human biology and genetics investigators can be required to master the basics of behavioral and social sciences.

NIDA already is sponsoring a series of training workshops for new investigators to expose them to the different disciplines that now contribute to its research mission. The initial workshops have focused on epidemiology, pharmacology, and neuroscience and introduced participants to those fields. Future workshops are planned, with a broad agenda that cuts across the behavioral and social sciences, including ethnography and behavior genetics, as well as domains of medical sciences such as proteomics, drug development, and NIDA's clinical trials network.

Sustained investment in research education of these types will be needed at NIH and NIDA. Without attention to pharmacology, neuroscience, and pharmacogenetics, it will be difficult for future investigators to develop a fundamental understanding of the pharmacological and economic-compulsive categories of offending in the Goldstein-Brownstein tripartite conceptual framework. Without grounding in the social sciences, it will be difficult for them to develop a fundamental understanding of the systemic categories.

There now are investigators who can bridge the gaps that appear as canyons between disciplines. Elliott's attempt to articulate his work with the NIH human genetics initiative provides one example. In a primate lab run by Steve Suomi at NIH, research on gene-environment interactions as substrates of aggressive behavior, social maladaptation, and drug use provides another example. This research is especially useful because the environmental conditions experimentally manipulated in this lab have conceptual linkages back to the deviancy training, inept parenting, and parent-infant relationship models developed by Patterson, Dishion, Brook, and others (Higley et al., 1996a, 1996b; Higley, Suomi, and Linnoila, 1996a, 1996b; Patterson, Dishion, and Yoerger, 2000; Dishion et al., 1996; Brook et al., 1996; Brook, Tseng, and Cohen, 1996).

More examples of this type of bridgework are emerging from the work of the research pioneers who try to keep pace with evolving contributions from the NIH Human Genome Project. The NIH-NIDA research agenda can be enriched by a technical report series that brings examples of this type to the community of investigators and research trainees.

### **Tension that involves approach or methods**

Review of the drugs-crime literature creates an opportunity for developing new insights about the sometimes different approaches and methods that have been developed in public health and criminal justice research work groups. For example, ethnography with small groups has expanded to almost large-sample ethnographic research that bears some resemblance to large-sample survey research, but in many ways is different. To an outsider, this expansion is a puzzle to be solved and has not yet been grasped. In the public health research domain, the original role of an ethnographer bore some resemblance to the role of the medical practitioner as a student of the natural history of disease. The original natural history studies were intensive case studies, with the doctor at the bedside of individual sick patients making careful systematic observations about this individual case and then that individual case, in the days when there might have been symptom palliation (e.g., cold cloths for fever), but no effective curative interventions to change the clinical course of disease. This has some resonance with Agar's concepts of

the ethnographer's attention to the details of behavior and verbal expression and of writing the narrative and taking down the stories of drug users in their own words (Agar, 1973). The link from this role of the ethnographer to large-sample ethnography remains unclear.

Measurement methods pioneered in behavioral sciences research and introduced to studies of drug taking by Larson, Kaplan, and Schiffman have started to surface in criminal justice research as well. Experience Sampling Methods (ESM), originally developed to study the daily lives of high school students, have now been introduced in research on drug use (e.g., see Csikzentmihalyi and Larson, 1987, 1992). Their ESM procedure requires study participants to wear an electronic pager device that beeps at randomly scheduled intervals, signaling the participant to record some predetermined aspects of his or her present feelings, activities, and/or surrounding environmental conditions. Usually, dozens of self-reports are collected over a week or more to capture as much of participants' daily living as possible. One advantage of this method is the ability of the researcher to examine drug use specific to each individual, given the assessment of his or her baseline characteristics for comparison. An additional benefit is the possibility of taking into account measured social context of the behavior (e.g., see Farnworth, 2000). ESM also creates new opportunities to investigate the determinants of drug-taking behavior that might be unique to each individual and each situation (e.g., see Kaplan and Lambert, 1995).

These evolving ESM procedures require a number of conditions if reliability and validity are to be enhanced. Kaplan and Lambert (1995) identified the following prerequisites: having a favorable and trusting relationship between study participants and researchers, ensuring complete confidentiality of responses, meeting labor or equipment and programming costs associated with beeping the participants several times per day, and addressing difficulties faced when the participants are illiterate or challenged by technology.

Several recent studies of delinquent and antisocial behavior may help clarify the utility of ESM procedures in research on the drugs-crime relationship. For example, Farnworth (2000) studied a group of young Australian offenders on probation and found that these respondents were engaged in such productive activities as employment or education an estimated 10 percent of the time. Compared with reference norms for Australian adolescents, offenders spent 30 percent more time on passive leisure activities. An estimated 42 percent of the time, offenders on probation reported being bored, while 62 percent of the time they were involved in unchallenging activities. The use of ESM to integrate studies of drug-taking and criminal behaviors will provide new and important evidence on relationships that generally have been studied via retrospective reconstruction of behavior over long spans of developmental time.

On another measurement front, there is a related tension that involves the use of bioassay methods to study recent and past drug taking. Wish has been a pioneer in the use of these methods for research on arrestees, and recent studies by Harrison and Fendrich are extending this reach into general household population samples of the type surveyed for

the National Household Survey on Drug Abuse (Wish, 1988; Yacoubian, Wish, and Perez, 2001; Fendrich, 2001; Harrison, 2001). In future research, one may anticipate these differences in approach to sustain a tension until a general consensus has evolved.

With respect to approach in the domain of statistical methodology, computational advances have contributed to an acceleration of innovation. There is a resulting air of optimism for what might be accomplished, as in the domains of longitudinal latent transition modeling, multilevel or nested models, approaches to nonignorable missing data, and alternative methods of research on directed acyclic pathways with mediation versus cyclical pathways with reciprocities. At the same time, there is a tension because these new statistical approaches have not become integrated in most research training programs, and there remains certain skepticism about heavily modeled data.

Limitations on numeracy keep many of us from probing the assumptions of complex models, whether these are models of behavior in individual studies, econometric models, or operations and systems research models to probe alternative program and policy decisions. Tension may be inevitable in the face of such complexities.

### **Tension involving research ethics**

NIJ can play an important role in relation to investigations that probe drugs-crime relationships. At present, a good part of the NIJ role has been ceded to HHS and its new Office of Human Research Protections (OHRP). True to its origins in NIH, OHRP is oriented toward the standards of experimental medical intervention research (e.g., randomized trials to test safety and efficacy of new drugs). OHRP specifications for informed consent procedures and disclosure statements share this orientation.

Many behavioral and social sciences researchers have expressed concern that the standards and specifications of experimental medical research are not appropriate for studies of the drugs-crime relationship. For example, in ethnographic and observational survey research, different specifications for informed consent procedures and disclosure statements are required.

NIJ officials can initiate a useful dialogue with OHRP on this important research topic. Perhaps more than any other government agency, NIJ can help to stimulate a dialogue and negotiate a reorientation of current practices in a manner that fosters new and creative research on the drugs-crime relationship without a lapse in research ethics or slippage in the protection of human subjects in this research.

Outside the Federal Government, researchers now face increasingly thorny challenges in the protection of their research participants and the assurance of confidentiality in relation to research data. For example, research that includes assessments of tobacco smoking now requires special handling as a result of legal action by the tobacco industry. These requirements apparently extend to criminal justice research in which tobacco smoking is approached as a self-reported indicator of deviance. The integration of molecular biology and genetics into these research agendas, and even the introduction of experience sampling methods or bioassays for drug testing, raise new questions in the domain of

research ethics, some of which have been scrutinized in randomized experimental designs. These challenges deserve the close attention of these research communities, with OHRP and its NIJ counterpart in suitable roles.

### **Does Drug Use Cause Crime? A Focal Point**

Each author of working papers for the drugs-crime research forum was asked to identify a circumscribed set of research issues and probe what we really know about them. Mindful of other sections to be written, we have been able to organize these research issues in relation to a single focal point, expressed in the question, “Does drug use cause crime?” One advantage of this specific focal point is that it has a broad range and can encompass many different strands of evidence developed in public health and criminal justice research. Another advantage is that it is a crucial open question for research on crime and drugs. As characterized by Harrison and Backenheimer (1998), “Research has not been able to validate a causal link between drug use and criminal behavior.”

When confronted with an etiological research question such as “Does drug use cause crime?,” a public health scientist typically might turn to a 20th-century elaboration of the 19th century Henle-Koch postulates or conditions for judging whether a specific disease might be caused by specific bacteria. For a time, this 20th-century elaboration was known as Hill’s postulates (after Sir Austin Bradford Hill, a medical statistician) and also as Evans’s postulates (after A.S. Evans, an epidemiologist; Evans, 1976; Hill, 1965). Today, students of epidemiology learn them as criteria for judging whether an association is causal or guidelines for evaluating the evidence of a causal relationship, together with an analysis of the relative strengths and weaknesses of evidence from randomized trials, prospective and longitudinal studies, retrospective studies, and case-control comparisons. Exhibit 8 presents these criteria and guidelines.

Before reviewing these criteria, four clarifications may be in order. First, the criteria for evaluating causal significance of observed associations represent standards of scientific evidence that are substantially different from the standards used to judge causal evidence in civil and criminal proceedings. For some segments of this paper’s readership, the question, “Does drug use cause crime?” may sound silly: “Of course drug use causes crime. My grandmother could tell you that.” (This was the type of reaction TV/radio personality Rush Limbaugh gave to some of the early work that Howard Chilcoat, Tom Dishion, and I published on the topic of whether inner-city mothers and fathers might be able to help protect their children against risk of early-onset drug use if they maintained levels of parental vigilance generally associated with good parental supervision and monitoring.)

Our response to these gentle readers is to beg forbearance. Of course, some of what our grandparents learned to be true is not true, and the analysis of responsibility for negligent or criminal acts in the individual case (as in a court of law) necessarily has a different set of standards of evidence. For example, evidence beyond a reasonable doubt is not the same as the definitive evidence referenced in the first paragraph of this paper.

Our second clarification is that we acknowledge a possibility that delinquent or criminal behavior might be a cause of drug use, the chicken-egg problem referenced by Inciardi and advanced with evidence by others. This possibility surfaces when one considers earlier sociological models of deviance (e.g., Sutherland, Matza) or later sociopsychological developmental models for youthful deviance, antisocial behavior, and delinquency, such as the coercive interaction and deviancy training models introduced by Patterson, Dishion, and colleagues; Coie and his colleagues at Duke; and Kaplan at Texas A&M University (e.g., see Patterson, Reid, and Dishion, 1992; Patterson, Dishion, and Yoerger, 2000; Dishion et al., 1996; Coie and Lenox, 1994; Sandstrom and Coie, 1999; Bagwell et al., 2000; Hubbard et al., 2001; Kaplan, 1995). For example, minor rule violations in early childhood, well before the years of starting drug use, might be followed by general peer rejection, differential association or affiliation with other rejected and deviant peers, and subsequent group-fostered delinquency and norm violations, including illegal drug use. We note that this possibility, and the more advanced idea of reciprocities between drug use and criminal behavior, do not necessarily undermine inferences about drug use as a cause of crime. We face a problem of slightly different conformation in our research on drug dependence: The use of a drug is an absolutely necessary but not sufficient condition for development of clinical syndromes of drug dependence, but once the drug dependence process has started, the drug dependence takes on a life of its own and becomes a determining influence for subsequent drug use (i.e., drug use causes drug dependence, and then drug dependence causes drug use).

Our third clarification is to ask first whether it is plausible that there is no association between drug use and crime or criminal behavior or whether there might be an inverse association (the more crime, the less drug use). In our review of available evidence, we must acknowledge the possibility that in some subsegments of human experience, there well may be a negative association between drug use and criminal behavior (e.g., in the highly disciplined and controlled environments of industrial espionage), just as we must acknowledge the fact that some 90-year-olds have smoked a pack or more of tobacco cigarettes virtually each day of adult life and have not developed lung cancer. We also acknowledge the high probability that in certain times and places or in certain subsegments of population experience, there is no association between drug use and criminal behavior (e.g., see Blum and Associates' studies of clinicians and professionals who used LSD before it was regulated by the Food and Drug Administration; Blum and Associates, 1964).

Notwithstanding these exceptional circumstances, there is a generally consistent overall pattern of positive and sometimes quite strong associations between illegal drug use and criminal behavior of other types. These associations are observed not only in samples of offenders in the criminal justice system (e.g., DUF and ADAM), but also in general household population samples. This evidence has some vulnerability due to constraints on methods (e.g., refusals by study participants to give informed consent for participation), but recent consistent evidence from general population surveys indicates that the observed association extends beyond officially recognized crimes and does not suffer the transition bias that is present in DUF, ADAM, and other criminal justice

samples (e.g., perhaps the arrested or incarcerated offenders were caught because of impairments from drug use, or the drug use of an offender is a manifestation of a more general characteristic of carelessness that might lead more readily to apprehension by the authorities).

Fourth, a “cloud of confusion” sometimes descends when people begin talking about causes and causation. We will try to be clear. Although we are asking whether drug use causes crime, we are not saying that there are no other causes of crime. This issue sometimes is subject to misinterpretation. For this reason, it might be more sensible to express the question in a different way: “Under what conditions, if any, does criminal behavior, as a response variable, depend in any substantive way on drug use, such that we might be able to shape criminal behavior by shaping drug use?” This question is not as pithy as, “Does drug use cause crime?” but it might help us escape the cloud of confusion when we try to review available evidence pertinent to this issue of causal inference.

### **Criterion/guideline 1: Temporal relationship**

If illegal drug use is believed to be a cause of criminal behavior, then we require evidence that illegal drug use has preceded the onset of that criminal behavior. Judgments about this criterion or guideline can become difficult when there are potential reciprocities. For example, when sustained medicinal use of phenacetin and acetaminophen compounds (e.g., Tylenol, Datril) was being investigated as a cause of interstitial nephritis and end-stage renal disease (ESRD), one of the complications was the possibility that the earliest clinical features of ESRD include headaches. Of course, headaches can promote the sustained use of pain-relieving medicines, including the acetaminophen compounds.

The drugs-crime relationship presents this type of temporal complexity, as was seen in exhibits 6 and 7. Earlier aggression, conduct problems, and criminal behavior may function as a direct cause of illegal drug use (e.g., see Kellam and Anthony, 1998), and possibly as an indirect cause (e.g., by promoting affiliation with other delinquent and drug-using peers). Earlier drug use also may function to promote later growth of conduct problems or criminal behavior (e.g., see Johnson et al., 1995).

### **Criterion/guideline 2: Biological or other theoretical plausibility**

Carrying books of matches is associated with the risk of developing lung cancer, tends to precede rather than follow the onset of lung cancer, and has at least a moderately strong association with lung cancer. However, except with respect to the associated characteristic of tobacco smoking, we have no biological or other theoretical plausibility to link carrying matches per se with the etiology of lung cancer. Even if the matches-cancer association were to withstand the challenges posed by the other criteria for evaluating causal significance of an association, we would be inclined to ask about the underlying theory and its plausibility and coherence in relation to known relationships and facts.

The tripartite model for the drugs-crime nexus represents a substantiation of plausible causal links from illegal drug use to criminal behavior. Other related strands in the fabric

of plausibility have been mentioned (e.g., differential crime opportunity, differential association).

The plausibility of a link between drug use and aggressive or violent crimes rests to some extent on neuroscience theory and observed clinico-pathological associations, as in contemporary thinking about cocaine's influence on limbic-hypothalamic substrates of aggression (Davis, 1996). In addition, there is a line of preclinical and clinical laboratory experiments that has helped to solidify the plausibility of a link from drug use to aggressive or violent behavior, and possibly to the types of norm violations associated with nonviolent crime. The evidence on links from the use of psychostimulant drugs (e.g., methamphetamine, cocaine) and aggression is noteworthy in this respect. Administration of cocaine to hamsters during adolescence increased the number of bites and attacks indicative of a surge of offensive aggression (Harrison et al., 2000). Moore and Thompson (1978), studying pigeons, found that high doses of cocaine elicited aggressive behavior. In some species, increased levels of aggression also have been observed with the administration of amphetamine stimulant drug—not only when a large single dose (e.g., Melega et al., 1997), but also after sustained lower doses (Haber, Barchas, and Barchas, 1981) are administered. These psychostimulants also may increase the risk of self-directed aggression (e.g., see Pepper-Smith et. al., 1983).

Experimental laboratory research with human subjects also has produced supportive evidence along these lines, often with computerized point-subtraction methods used to evoke aggression after the drug has been administered and under control (no drug) conditions. For example, Licata et al. (1993) administered a high dose, low dose, and no dose of cocaine and found that subjects in the high-dose group expressed significantly greater aggression than subjects in the control group; the low-dose group did not differ from the control group.

Notwithstanding these strands of plausibility, there also is a considerable amount of inconsistency in the observed data and some complexity in relation to dose-response analyses. For example, Crowley et al. (1992) found no increase in aggression when cocaine was administered in primate lab research; Darmani and colleagues (1990) found increased aggression among mice that were given relatively low doses of cocaine but not when the mice were given higher doses of cocaine. Moro et al. (1997) found reductions in the total number and length of aggressive activities in mice after amphetamine administration. Cherek et al. (1989), studying humans, examined the relationship between d-amphetamine on aggression using point subtractions and found an increase in aggression among those receiving 10 mg per 70 kg of body weight but a decrease in aggression when 20 mg per 70 kg of body weight was administered.

Police experience on the street implicates dissociative drugs such as phencyclidine (PCP) in relation to violent and aggressive behavior and crime. We have been able to find some supportive experimental laboratory evidence consistent with this streetwise experience (e.g., Burkhalter and Balster, 1979; McCardle and Fishbein, 1989). Nevertheless, even with PCP, there is a complex pattern of inconsistent evidence that does not ring true with the experience on the street and common wisdom about PCP. Tyler and Miczek (1982),

Emley and Hutchinson (1983), and Miczek and Haney (1994) reported no increase in aggressive behavior after experimental administration of PCP and an erratic increase in aggression only in a subgroup of animals receiving low doses. Hence, it may be that PCP promotes aggression only in certain subgroups of the population (e.g., see McCardle and Fishbein, 1989); and in some experiments, animals receiving high doses of PCP were more likely to be victims of aggression by nondrugged animals (e.g., see Russell, Greenberg, and Segal, 1984; Tyler and Miczek, 1982).

In sum, there is some plausibility to the idea that drug use might promote criminal behavior, with strands of plausibility coming from neuroscience theory, the common wisdom and experience of criminal justice officials and drug users, and laboratory experiments. The links between being a drug user and becoming a crime victim represent an understudied phenomenon, and the inconsistent patterns of laboratory evidence provoke us to investigate the possibility that there might be substantial heterogeneity within the population with respect to links from drug use to aggressive behavior or to crime (e.g., see Parker and Rebhun, 1995).

History demonstrates one of the difficulties with this criterion for judging causal significance of associations. Time and time again, new evidence has contradicted what appeared to be a biologically plausible or theoretically pleasing link between a suspected cause and a suspected response. Today's biologically plausible or theory-driven causal inference may be tomorrow's "old wives' tale." As is true for the other criteria and guidelines, by itself this one counts for little.

### **Criterion/guideline 3: Consistency of the association**

We already have clarified the possibility that no association or a negative association might exist for certain subsegments of population experience. For example, at some point, drug taking may incapacitate an individual who otherwise would be involved in criminal behavior. Despite examples of this type, and notwithstanding contrary evidence, the drugs-crime research literature now includes a generally consistent replication of positive associations between illegal drug use and criminal behavior (e.g., see Harrison and Backenheimer, 1998).

The body of laboratory experiments on drugs and aggressive or violent behavior is not as consistent as one might expect. As described under criterion/guideline 2, under some circumstances, laboratory experiments have established a small set of drugs as causal agents in relation to aggression and violence. However, for most drugs and many circumstances, there are negative findings, and the evidence is not consistent with causal links from drug taking to aggressive and violent behavior.

Given the multiplicity of drugs, types of crimes, and varieties of social contexts, it may be inevitable that the accumulated body of evidence on the drugs-crime relationship appears inconsistent. Variation in the quality of the research also has a bearing on consistencies or inconsistencies in the evidence. As every first-year graduate student learns, research with imprecise measurements will tend to yield evidence of no

relationship even when a relationship exists; research with measurements of limited validity will tend to yield evidence of relationships where none exists.

Although not generally introduced as a feature of studying consistency of relationships between causes and effects, a developmental perspective may help to lead the reader to a greater appreciation of inconsistencies and complexities faced in research on the drugs-crime relationship. That is, the timing of the onset of the drug taking may condition the later expression of criminal behavior and may lead to greater heterogeneity in the population with respect to the drugs-crime relationship. For example, we have some evidence on the possibility that earlier-onset drug use is associated with later risk of developing drug problems (e.g., see Anthony and Petronis, 1995). We also have evidence that prompts us to conceptualize earlier-onset drug use as a type of precocious adolescent development that may disrupt normative developmental trajectories (e.g., see Newcomb, 1992; Dawes et al., 2000). There may be a tendency to interpret these disruptions as sources of increased levels of later criminal behavior, consistent with the idea that risk of drug problems are increased for early-onset drug users; this has been the perspective our research group has taken in its studies of this topic (e.g., Johnson et al., 1995; Anthony and Petronis, 1995). Nonetheless, it is possible that precocious (i.e., early onset) drug taking is followed by disproportionately greater increases in frequency of drug use and in risk of drug problems but that the early-onset drug use dampens the level of criminal behavior that otherwise might occur if the drug use had not started so early.

Our study of early-onset alcohol use and the later developmental trajectory of conduct problems represents a case in point. In that study, cited above under criterion/guideline 1 (Johnson et al., 1995), we found that baseline levels of conduct problems were greater for boys who had started drinking alcohol before the adolescent years without parental permission and that growth of conduct problems was greater for these early-onset alcohol users—*when compared with boys whose drinking did not start until later*. Similar relationships were observed for girls with early-onset alcohol use—*when compared with girls whose drinking did not start until later*. However, a discussion of this research with Blumstein has prompted us to re-approach this problem with a different comparison in mind. Using random effects regression, we are seeking to hold constant the baseline level of conduct problems and study boys who have a high initial level of conduct problems but who start drinking alcohol early on and compared them with boys with an equally high initial level of conduct problems but for whom alcohol consumption is delayed until adolescence. Approaching the contrast in this manner, we may discover that early-onset drinking dampens the growth trajectory for conduct problems; the steepest trajectory for growth of conduct problems may be observed for boys with high initial levels of conduct problems but without the impairments associated with early drinking. The early drinking might lead to retardation in the growth of conduct problems for boys who otherwise would escalate to very high levels of conduct problems in adolescence.

This is a somewhat counterintuitive proposition, and it may run counter to common wisdom and experience with respect to the effects of early-onset drinking or drug use and the later lifecourse of young people. However, our intuitions and common experience about these circumstances tend to reflect a type of population-averaged summary of

developmental trajectories and generally do not encompass all varieties of human experience. We mention this open research question as an example of the complexities faced in developmental research on the drugs-crime relationship and as a possible explanation for the inconsistencies observed in drugs-crime research. The timing of the drug use may induce subgroup variation in the drugs-crime relationship, which then is interpreted as inconsistency in and a challenge to causal significance of the observed associations.

Fortunately, complete consistency of evidence is not required. What is required is a focused probing of the circumstances under which the drugs-crime relationship is a causal relationship, with a deliberate effort to ferret out situations in which there is no causal linkage between drug use and criminal behavior. Deliberate scientific pursuit of these circumstances and situations may require investigators to look overseas, where use of such drugs as marijuana, cocaine, and heroin are not treated as criminal behaviors. In social contexts of this type, by studying the developmental trajectories of criminal behavior among young people with and without early drug-taking experiences, we may be able to illuminate some of the inconsistencies now observed in the drugs-crime evidence available to us. For example, longitudinal studies of children growing up in the Netherlands are underway. The recent effective decriminalization of marijuana use in the Netherlands creates a social context for research on this drug and later criminal behavior that merits attention on the NIH-NIDA research agenda.

**Criterion/guideline 4: Alternative explanations ruled out**

This criterion or guideline represents the Achilles heel for much of the prior research on the possible causal links between illegal drug use and criminal behavior and represents a general difficulty for observational research in general. Observing a possible causal relationship between antecedent A and response B, the skeptical critic always can ask, “Isn’t there some unrecognized background factor C that can account for the A-B relationship that you have observed in this study?” If so, “Isn’t this a poorly developed conceptual model?”

To some extent, these are a coward’s questions about the drugs-crime relationship in specific and about empirical research in general. Of course, there might be some unrecognized background factor in empirical research plans and in completed studies, if not the hand of the mischievous Norse god Loki, then something else of a less celestial nature.

The challenge for the courageous skeptical critic is to assert a specific background factor or set of background factors that might account for the observed A-B relationship and that have not been considered explicitly or taken into account in a study plan or description of completed work. For example, observing the suspected causal association between tobacco smoking and risk of lung cancer, the statistician Sir Ronald Fisher posed a question of the following type: “Can’t we explain the observed association as a manifestation of an underlying predisposition or liability that determines both the tobacco smoking and the lung cancer?”

In relation to the drugs-crime relationship, the most plausible background factors seem to be of the variety named by Fisher, namely, unmeasured predispositions; in this instance, the predispositions might involve who abides by the conventional rules of society, who is willing to run afoul of the law by taking a drug illegally, and who is willing to commit crimes other than the crime of drug possession for personal use. To some extent, these predispositions may be a manifestation of family heritage, a manifestation of early experiential conditions and processes, or a synthesis of both. Nevertheless, no matter what their origin, until these predispositions are taken into account, they represent a plausible form of alternative explanation whenever a drugs-crime relationship is found in our empirical studies.

One line of response to this criticism has been to measure personality or facets of temperament in observational studies and to re-estimate the drugs-crime association with personality or temperament held constant (e.g., via stratification or statistical adjustment in a regression model). But this response always is subject to the criticism that the wrong facets of personality or temperament were measured or that the measurement of personality or temperament was not as good as it should have been.

It is in relation to this criterion that we now have new opportunities for research at the intersection of public health and criminal justice research on the drugs-crime relationship. Three important opportunities at this intersection involve (1) genetics, twin, and family research; (2) longitudinal studies with “subjects as their own controls” designs; and (3) controlled experimental trials.

Future genetics, twin, and family studies can help to narrow the alternative explanations in a useful manner. For example, in an earlier section we described a design that exploits the genetic matching of monozygotic twins to search for environmental conditions that contribute to the occurrence of disease. Discordant MZ twin designs also can be used to hold constant predispositions or liabilities linked to the individual genome of the twins, while studying differences in the trajectory of criminal behavior for the MZ twin whose illegal drug use starts first versus the MZ twin whose illegal drug use starts later (or not at all).

Alternative twin and family research designs can be used to narrow other explanations of the observed drugs-crime relationship (e.g., studies of discordant siblings, studies based on the transmission disequilibrium test when specific polymorphisms are under investigations). Cadoret and colleagues have offered recent illustrations of the power of twin studies in which some twins have been separated at birth, but these “natural experiments” have become scarce in the United States and other parts of the world where twins now generally are kept together in their new adoptive families (e.g., see Cadoret et al., 1986, 1995; Cadoret, Leve, and Devor, 1997). Tsuang et al. (2001) provide a recent useful overview of pertinent findings from the Harvard Twin Study.

Longitudinal subjects-as-their-own-controls research designs also can help rule out alternative explanations in the sense that each individual participant is carrying forward a within-individual set of propensities to become engaged in illegal drug use and other

criminal offending. In these longitudinal designs, in an otherwise law-abiding individual, if we were to observe that criminal offending occurs only in the immediate aftermath of a drug intoxication experience or only in the stages of withdrawal after drug dependence, we would have additional evidence of a drugs-crime association at the individual level. These longitudinal designs remain vulnerable to a possible counterclaim that there is an underlying predisposition that links earlier illegal drug use to later criminal offending only during the context of drug intoxication or withdrawal states. That is, the observed association between illegal drug intoxication or withdrawal and the later criminal offending is a spurious artifact of uncontrolled confounding: There is something else in the background, a third variable that explains the observed sequence.

Medical and public health research is host to a variety of examples of this type of spurious confounding. One of them involves the connection between chickenpox and shingles. For most people, chickenpox occurs early in life and shingles occurs late in life. There sometimes is an exceptional case of shingles occurring with no prior history of chickenpox in childhood, but these exceptional cases might be understood as instances of “clinically inapparent” infections (i.e., with mild or minimal symptoms in childhood, so mild as to pass without notice). A longitudinal research design on this topic can lead to the impression that chickenpox causes shingles, in the sense that shingles rarely or never occurs unless chickenpox occurs first. This observed longitudinal link between chickenpox and shingles satisfies the requirement described above: Criminal behavior occurs only after a bout of illegal drug use. The fly in the ointment in our chickenpox-shingles example is that we now know that chickenpox does not cause shingles. Rather, it is an underlying virus that causes both of these clinical phenomena. Exposure to the chickenpox virus (herpes zoster) is the cause of the chickenpox in childhood and is the cause of shingles in later life when the virus emerges from an otherwise dormant or latent state of no activity. The apparent linkage from earlier chickenpox to later shingles is due to an underlying third variable, the herpes zoster infection, which accounts for the appearance of both outcomes.

The analogy to research on illegal drug use and later criminal offending should be clear. Even when longitudinal research shows us examples of participants who become engaged in criminal behavior only in the context of drug intoxication or withdrawal states, we cannot be confident that the illegal drug use is the cause of the associated criminal offending. Some unknown underlying cause may be accounting for both outcomes.

The third approach, involving randomized trials, offers a way to bring these unknown underlying variables into check. This approach already has been described in relation to our research group’s studies of an alternative explanation for the drugs-crime relationship. Namely, we advanced the hypothesis that a predisposing characteristic in the form of early aggression or rule breaking is a potentially modifiable determinant of both later illegal drug use and criminal behavior or other sorts. This hypothesis does not reject the possibility that illegal drug use causes later criminal behavior, but it introduces one alternative explanation for the observations association between illegal drug use and criminal behavior (i.e., the earlier aggression or tendency to break rules and social norms). As described earlier, we sought to test this hypothesis by constructing an

experimental trial in which we disrupted the development of early aggression and rule breaking (e.g., Kellam and Anthony, 1998). We used the power of randomization to hold constant the profile of alternative explanations that might account for later illegal drug use and criminal behavior. In a current followup study of the youths who participating in this trial, we will be testing whether the primary school intervention had a sustained impact on illegal drug use and criminal behavior. If so, we might expect a weakened association between illegal drug use and criminal behavior in the subgroup of youths exposed to the active behavioral intervention arms of our study.

A related opportunity to test the drugs-crime relationship and to use randomization to rule out alternative explanations involves controlled trials of new therapeutic interventions directed toward illegal drug use and drug dependence of adolescents. Observational studies now suggest that entry into drug treatment reduces the rate of criminal offending, but these studies leave open possibilities for alternative explanations (e.g., selection biases in the assignment of subjects to treatment, imbalances in the other determinants of criminal offending). Randomization in the setting of controlled trials of new therapeutic interventions creates an opportunity to constrain these selection biases and bring into balance the alternative sources of variation in criminal offending (e.g., see Manski et al., 2001).

By adding followup measurements of posttreatment criminal behavior to current and newly emerging randomized controlled trials of therapeutic interventions, NIJ and NIDA can help foster new evidence on the degree to which illegal drug use is a cause of criminal offending. Alternative explanations for the observed drugs-crime association and other determinants of the offending behavior can either be brought into balance by randomization or held constant as measured covariates in statistical models of analysis. Some examples of past research along these lines are described under criterion/guideline 7.

#### **Criterion/guideline 5: Dose-response or gradient relationship**

Absence of a dose-response or gradient relationship does not rule out causal associations; there are good examples of threshold relationships with no clear gradient. Nonetheless, there are examples in which the probability or rate of criminal behavior is observed to be lower with lower levels or frequencies of illegal drug use and is observed to be greater as levels or frequencies increase.

In one recent and especially informative longitudinal cohort study, Brook et al. (2001) studied the developmental trajectory of marijuana use from childhood into adulthood and found that behavioral and attitudinal indicators of unconventionality (e.g., attitudes tolerant of norm violations) had a gradient relationship with later increases in marijuana involvement. The research team also found that as levels of unconventionality increased, so did marijuana involvement. These gradient relationships between unconventionality and marijuana use help to substantiate a possible causal link between earlier unconventionality and later developmental trajectories of marijuana involvement. However, as in the circumstance of research on the drugs-crime relationship, this research report leaves us with unanswered questions of the following variety:

- a. What about the predisposition that links unconventionality to the earliest marijuana use? Where does the unconventionality come from, and is this predisposition the same as the predisposition to smoke marijuana?
- b. What about the reverse causal pathway and the possibility that increasing marijuana use might promote later increases in unconventionality?
- c. As levels of marijuana use increase, are there later dose-response or gradient-like increases in unconventionality?

In light of the population heterogeneity mentioned above, this dose-response criterion might be especially troublesome in research on the drugs-crime relationship. For example, consider the drug user whose increasing bouts of intoxication yield less criminal behavior than otherwise might occur and whose intoxication-associated carelessness leads to apprehension and detoxification and outpatient treatment prior to a bench appearance. The detoxification and treatment might be followed by a return to the baseline level of criminal behavior (i.e., a higher level of criminal behavior than was observed during the period of intoxication) and an impression that treatment was ineffectual with respect to the frequency of criminal behavior.

#### **Criterion/guideline 6: Strength of association**

Weak associations seem especially vulnerable to sources of spuriousness and bias. One benchmark standard for strength is the association between tobacco smoking and lung cancer: The risk of dying from lung cancer is estimated to be 10 times or greater for persistent tobacco smokers than for nonsmokers. Toward the other end of the spectrum of magnitude is a widely appreciated but quite modest strength of association between being male and illegal use of drugs: The risk of becoming an illegal drug user is an estimated 1.5 to 3.0 times greater for an American male than for an American female (Anthony and Helzer, 1995).

Examining the range of study estimates on the drugs-crime relationship, there are some studies with extremely large relationships, but when a positive relationship is observed, the strength of relationship tends to be quite modest. This generally modest relationship may imply that alternative explanations (e.g., predispositions) are sufficient to account for the observed relationship.

#### **Criterion/guideline 7: Cessation effects**

Cessation effects already have been mentioned in the context of our discussion of alternative explanations under criterion/guideline 4. There are many studies of co-occurring maturation processes that lead to fading of both illegal drug use and other criminal behavior, especially since the work of Winick. The observational studies of McGlothlin, Anglin, and Hser in California and the work of Nurco, Lerner, and colleagues in Baltimore also shed light on declines in criminal behavior during periods of abstinence or reduced illegal drug use. The literature includes numerous studies of what

has happened to crime involvement after cessation of drug use, based on observational studies.

As noted under criterion/guideline 4, some of the strongest evidence about cessation effects can come from randomized experiments in which drug treatment or other interventions are used to disrupt illegal drug use, with subsequent evaluation of crime as an outcome of treatment. As noted under criterion/guideline 6, for some segments of the drug-using population, the cessation of drug use is followed by increases in frequency of criminal behavior (i.e., once impairments associated with intoxication are reduced).

**Reprise: Does drug use cause crime? What do we not know?**

This review of a specific hypothesized causal relationship was intended to highlight some of what we know about the drugs-crime relationship. Its main purpose was to provoke discussion and help in a process of identifying weaknesses and gaps in evidence that might be used to guide a future research agenda.

Evaluated in relation to these conventional criteria or guidelines for judging the causal significance of observed associations, the reader may have a better appreciation for the uncertainty conveyed in a recent summary statement cited above: “Research has not been able to validate a causal link between drug use and criminal behavior” (Harrison and Backenheimer, 1998). The available evidence is ambiguous with respect to temporal relationships.

Instead, we offer a series of discussion points about what we might not yet know.

**Is the evidence on a temporal relationship compelling?** Illegal drug use precedes formal criminal behavior in some of these studies, but what about the earlier antecedents of both drug use and crime in the form of rule breaking, misbehavior, and minor norm violations? One can imagine a co-occurrence process that begins with expression of irritable temperament or aggression in the preschool years, followed by rule breaking or norm violations in the primary school years, and then later co-occurrence of illegal drug use and delinquent or criminal offending. Our own research group and others have added some evidence on the possibility that drug taking that starts by age 11 might promote growth trajectories for later conduct problems among both boys and girls. The pattern of co-occurrence of conduct problems and drug use is a centerpiece of Jessor’s problem behavior theory, and there is reason to look to experiments that will help us differentiate these problem behaviors (e.g., differential response of each form of problem behavior to different interventions, as suggested in Dishion’s early Adolescent Transitions experiment).

**Plausibility?** Our focus has been oriented toward the individual, but there is a perspective on the drugs-crime relationship that is more ecological or contextual in orientation. For example, a social environment characterized by illegal drug use of individuals might give rise to norm violations and criminal offending of other sorts, and not necessarily in the form of offending by the drug users but rather in the form of offending by others. The mugging of a heavily intoxicated drug user by a group of

nonusing passersby serves as one example of aggregate effects of illegal drug use on crime that would not be apparent in individual-oriented studies but would require multilevel studies of interrelationships between individuals.

**Consistency?** What about the exceptions to a general pattern of observations? It seems likely that the drugs-crime association varies from time to time, place to place, and subgroup to subgroup. The study of variation in these patterns of association will help to disclose the boundary conditions and mechanisms that give rise to strong, weak, and possibly inverse associations. Research across borders and in settings such as the Amsterdam cannabis environment can help illuminate these boundary conditions.

**Alternative explanations?** Several lines of research have been started on the common causes for both illegal drug use and other criminal behavior, some of them originating in family history studies and the clever adoption paradigm adapted by Cadoret and his colleagues, some with a sharper focus on mechanisms of inheritance (e.g., assays of genetic polymorphisms), and some with a focus on personality and early social environment. It is not clear that studies to date have provided adequate control over these sources of co-variation. Nonetheless, the longitudinal study of individuals over time has provided evidence from subjects-as-their-own-controls designs, and the randomized trials of interventions provide some evidence that, despite common causes, an intervention directed toward illegal drug use can reduce frequency of criminal behavior. Even if there are common causes (e.g., inherited traits), for many observers, the longitudinal evidence coupled with experimental evidence is sufficient to draw the inference that illegal drug use causes criminal behavior. Reasonable people will disagree about this inference from available observations, and the points of disagreement will lead us to specific experiments or new studies to gather evidence that will be more compelling.

**Gradient?** Is it possible that some of the inconsistency in observations about the drugs-crime relationship can be traced to (a) selective attention either to the lower end of drug involvement (e.g., among children, adolescents, or high school seniors followed through the college years; see Schulenberg et al., 1994) or to the higher end (e.g., among arrestees or clients in drug treatment programs); (b) possible thresholds in the gradient relationship, with between-sample heterogeneity with respect to the effective threshold; or (c) an uncertain metric for assessing the type or level of drug involvement? As described in the prior section entitled “Criterion/guideline 2: Biological or other theoretical plausibility,” we have noted some inconsistent pharmacological effects across dosage levels of the same drug and across different drugs. If we are to appropriately sort the consistent and inconsistent findings of field studies on the drugs-crime relationships, it may be necessary to reach for greater specificity with respect to dosage levels or intensity of drug use and also with respect to the pharmacological differences observed in laboratory experiments. It no longer is enough to sort drugs into the non-scientific colloquial “soft” and “hard” categories, nor to lump all “illegal drug use” as if there were no heterogeneities of effect across the various forms of internationally regulated drugs. The best field studies of the 21st century will abandon these relatively crude classifications and will not carry forward an obsolete tradition from the earlier ground-breaking days of drugs-crime research.

**Strength of association?** Due to uncertainties about reciprocal and dynamic interrelationships between drugs and crime, it would be advantageous to look closely at studies with fine-grained temporal analysis of the drugs-crime relationship and to estimate strength of association prospectively. This should be done in a manner that allows change in the level of criminal behavior to be gauged in relation to change in the level of drug use and vice versa, or with an expression of the relative risk of criminal acts with and without antecedent illegal drug use.

**Cessation?** Our recent National Research Council committee expressed concern that selection effects, transition biases, or other artifacts might lead to a spurious inference that criminal behavior declines or stops when illicit drug use is ended, either with or without intervention. The evidence on this criterion might require special scrutiny in light of concerns such as these.

## Conclusion

In the final section of this working paper, I would like to integrate the organizing conceptual framework presented in the section on the rubrics with the ecological concept of scale described previously. Here, there is an adaptation of the formal ecological concept of scale that includes the microcosm and an extension of the concept that reaches to the macrocosm of the international regulatory environment.

The integration of the five rubrics and the concept of scale is depicted in exhibit 9. The result is a two-dimensional grid with the rubrics on one axis and scale on the other axis and showing the conceptual domain where research on drugs and research on criminal offending intersect. Each rubric-scale intersection or subunit in the grid can be populated by past and current examples of research on the drugs-crime relationship. In some subunits, density of past and current research is quite high; work in these domains may require strengthening, or perhaps these investigators should be left alone to do their work. In other subunits, we have done little or no past research activity; these subunits might warrant attention in a new agenda for drugs-crime research.

Starting in the upper left-hand corner of this framework, we have the intersection of quantity research with the microcosm represented by the genes we inherit from our forebears. We may expect one day to have an investigation that produces quantitative estimates of the frequency of homozygotes and heterozygotes with respect to genes that are implicated in the drugs-crime relationship, just as we now have these estimates for the frequency of alleles mapped to apolipoprotein E4 and other genes or polymorphisms implicated in the risk of developing Alzheimer's disease.

Working our way to the far upper right-hand corner, we stop at the level of nations. To the best of my knowledge, we have a limited set of quantitative estimates for rates of drug-taking behaviors and criminal justice statistics at the level of nations; but definitive evidence on variation across regions of the globe is lacking and represents a current gap in knowledge. To some extent, this gap can be filled by cross-national studies now

underway, such as the World Health Organization's (WHO's) recent World Mental Health 2000 research initiative being led by Ron Kessler at Harvard and T. Bedirhan Ustun at WHO, with collaborators in more than 20 countries around the globe (Kessler, 1999); the European School Survey Project on Alcohol and Drugs ([www.ipdt.pt/investigacao/espada99/indice.htm](http://www.ipdt.pt/investigacao/espada99/indice.htm)); and cross-national studies supported by NIDA in Latin America (e.g., Brook et al., 2001), including our own PACARDO Project (Anthony, 2000).

In the middle range, between the microcosm of the gene and the macrocosm of global regions, we have collections of estimates for various social and geopolitical groups. In aggregate, these estimates can help us to draw generalizations about the relative magnitude of problems associated with drug taking of one sort or another (e.g., marijuana use versus cocaine use) or with criminal behavior of one sort or another (e.g., aggravated assault versus shoplifting or vandalism).

Even within the rubric of quantity, there are many gaps. For example, our quantitative estimates often are based strictly on officially recognized offending and do not encompass unrecognized offending. With respect to drug taking, there is a plethora of evidence on the prevalence of drug use and drug dependence but not much evidence on the incidence or risk of becoming drug dependent. Here, also, we have big gaps in the evidence that warrant some attention as we design an agenda for future research.

The intersections of the location, causes, and mechanisms rubrics with the scale dimension brings us closer to evidence on variation from place to place, time to time, or in relation to personal characteristics. A few investigators have started to integrate genetic variation in their studies on such topics as drugs and crime, and soon we may have more definitive evidence on the relative frequency of different polymorphisms or gene-encoded protein products for different subgroups of the population or in different geopolitical zones. We can expect ecological analyses of the between-subgroup and between-zone rates, with new evidence on location.

Similarly, working outward from the simplest gene products to more complex products of gene-environment interaction, the sex hormones research of separate research groups led by Logan and by Angold, Costello, and others should provide us with more evidence on rates of antisocial behavior, drug use, and offending in relation to levels of testosterone and other hormones before and after drug use (Logan, 2001; Federman et al., 1997). The initial evidence is not expected to allow causal inference, but the understanding of locational variation will allow us to sharpen our causal theories and to integrate new biological perspectives on the drugs-crime relationship.

In a middle position in this framework, somewhat overlapping the different segments, the important line of research being conducted by Higley, Suomi, and their colleagues in relation to gene-environment interactions merits attention. This research, already mentioned in one of the preceding sections, touches on aggressive behavior, social conditions of child rearing, and drug use. Using a primate model, this research group has been able to extend the line of research on infant-mother relations that Harlow initiated.

The group is engaged in experimental manipulation of the early conditions of infant rearing, crossed with genetic predispositions that in the wild have been found to be related to aggressive behavior and excess mortality. The evidence from this research serves as an important example of how the effects of an apparently noxious inherited predisposition might be modulated by a change in child-rearing environments. Does this animal model of gene-environment interaction also hold for aggressive children, with later implications for their drug-taking behavior? Questions such as this one merit discussion in relation to the proposed drugs-crime research agenda, if only to choose not to pursue these lines of research.

Turning to the last row of the framework, I offer some speculations about gaps in research on prevention and control. At the level of scale that reaches from microcosm to the whole organism, I see a gap in research on underlying brain structures that subserve neuropsychological functioning of clear importance in the choice behavior of drug users and offenders. To the extent that drug users and offenders are making choices about various elements in their behavioral repertoires, we may be able to understand variations in response to prevention and control interventions as a function of neuropsychological performance (e.g., with respect to direction, control, and planning). Our comprehension of this variation can increase through a program of research on fMRI brain imaging and neuropsychological testing under experimentally controlled paradigms (e.g., aggression evoked through computerized point subtraction or other procedures). In time, we should be able to evaluate the degree to which response to these interventions depends on brain structure and function as manifest in neuropsychological tests as well as in response to genetic predispositions of the type now being characterized in Suomi's primate laboratory and elsewhere.

Working our way to the bottom right-hand side of this matrix, we find the intersections with social groups and contexts of increasingly larger scale, not only the peer group and family of origin or procreation, but also the larger neighborhood, employment context, the community at large, and across national boundaries. As we plot examples of intervention research in this two-dimensional framework, it is easier to find examples of individual investigations with narrow breadth of scale. For example, we can find an intervention focused on the community but without elements of intervention directed toward specific individuals in the community. We can find many interventions directed toward individual arrestees but not toward the social groups of which the arrestees are members.

One of the challenges for those who seek to shape the future research agenda on drugs and crime will be to encourage broadband research that cuts across multiple levels of scale. This is not to say that we should eliminate narrow-band research because it often is necessary to solve a research problem through focus, and focus is one of the defining characteristics of narrow-band research. Nonetheless, as we look over some of the more exciting research projects now underway, we can see that the excitement is coming from the investigators' attempts to encompass more than one level of scale. These attempts deserve encouragement.

Before closing, we must turn to the empty spaces created in the circle but not included as part of the two-dimensional grid. Within these spaces, we have important drugs-crime research that does not fall neatly into the two-dimensional conceptual framework. I am thinking of the recent ethnographic studies of the gangs involved in drug sales (e.g., Levitt and Venkatesh, 1998), and some of the other recent innovative qualitative research on drug trafficking (e.g., Natarajan, Clarke, and Belanger, 1996; Natarajan and Belanger, 1998), which shed new light on the structure and organization of the criminal organizations that sustain drug supply and influence drug-related criminal offending around the world. There also are good recent examples of operations research focused on the organization and administration of criminal justice agencies and the deployment of law enforcement, prosecution, and judicial resources (e.g., see Maltz, 1996). To the extent that these investigations guide us toward useful evidence about prevention and control, and to the extent that they focus on individuals or small groups of individuals (e.g., in a city or State), they may be placed in the space on the left-hand side of the figure, between the grid and the surrounding circle. To the extent that these control-oriented investigations are directed toward international drug trafficking (e.g., see Montagne, 1990), they may be placed in the space on the right-hand side of the figure.

There are other research programs and initiatives that do not fall neatly within the two-dimensional grid presented in exhibit 9. Methodological research constitutes one set of examples (e.g., Wish et al., 2000; Harrison, 1997, 2001; Fendrich, 2001). Proposed new research on drug prices and a consumer product index for illegal drugs represents another set (e.g., see Manski et al., 2001).

As we move toward a new drugs-crime research agenda for NIJ and NIDA at the intersection of public health and criminal justice studies, it is important to remember the two major themes mentioned in the introduction to this paper:

- There is no single drugs-crime relationship. Rather, there are drugs-crime relationships, most of which are complex rather than simple.
- There is no simple solution to the complex challenges faced when drugs-crime relationships come into play.

The two-dimensional grid encircled in exhibit 9 offers no simple solutions to the complex challenges faced when drugs-crime relationships come into play. That grid is only a tool that may help us identify important gaps in the research evidence, gaps that must be filled as we work toward a more complete understanding of the drugs-crime relationship and more effective action plans that apply new understanding in the service of public health and safety. In an important sense, the empty spaces encircling the two-dimensional grid also can be useful tools as we try to identify and fill the gaps in evidence. These empty spaces can serve to remind us that no conceptual framework is all encompassing. We must “think outside the box” in this regard. If we organize our scientific resources simply to continue our current lines of research, we will not achieve lasting reductions in illegal drug use and drug-related crimes, and we will never lay claim to great victories in the service of public health and safety.

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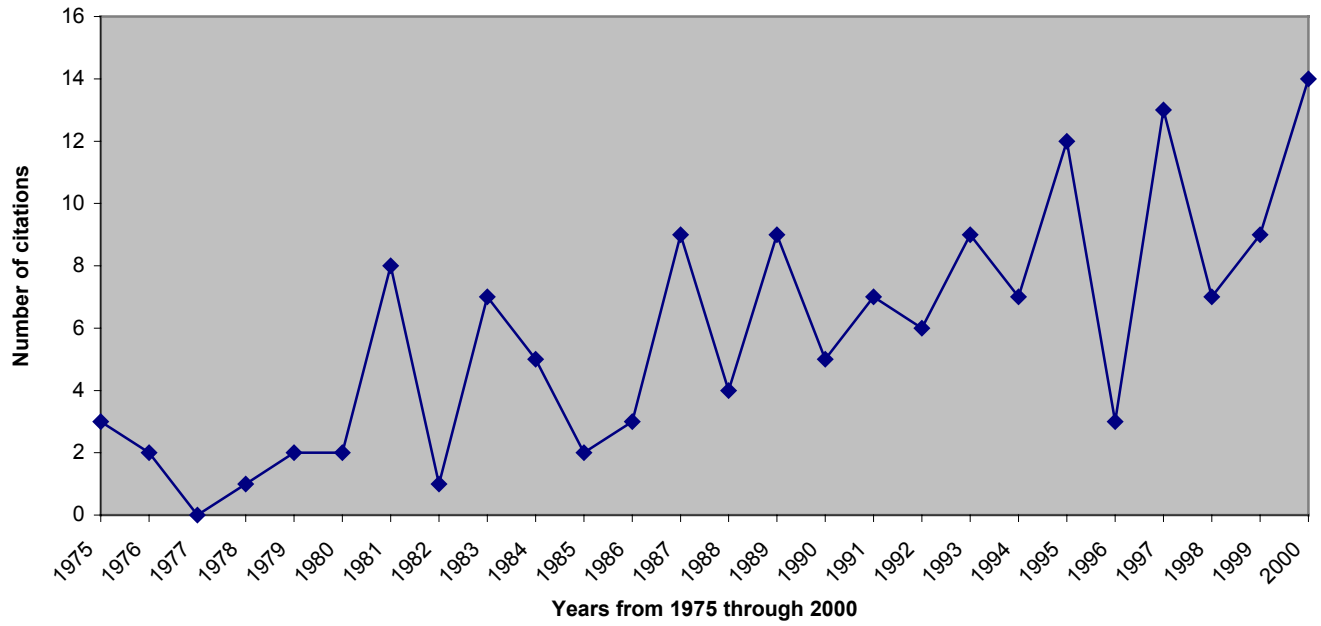
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**Exhibit 1. Number of Medline Citations: "Drugs & Crime"**



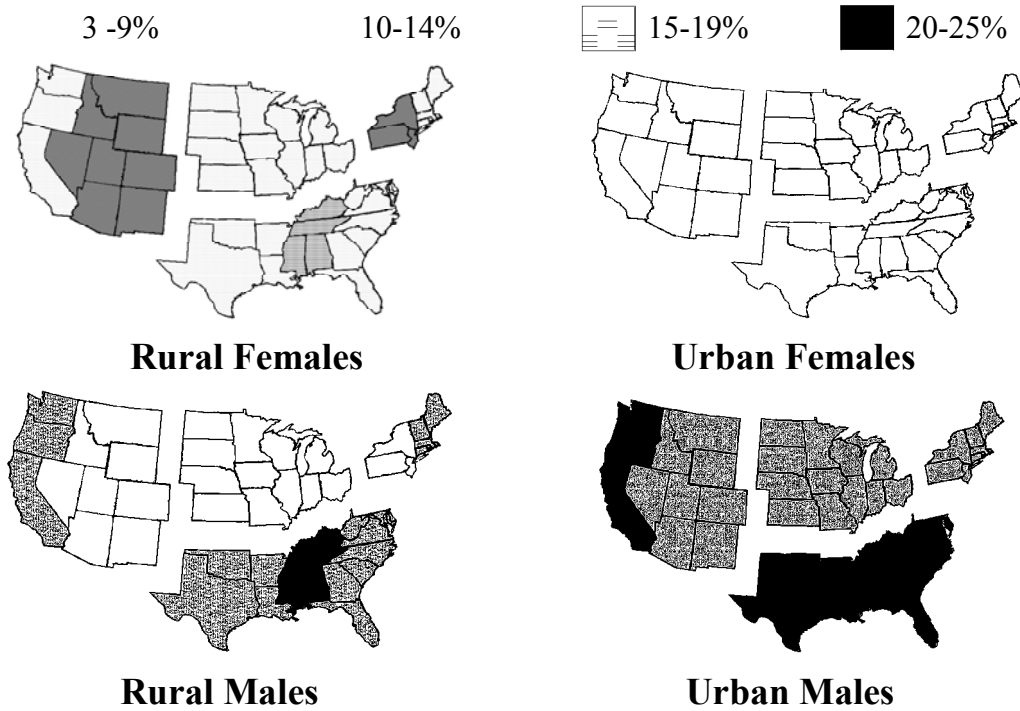
## Exhibit 2. The Rubrics of Epidemiology

Main Rubrics	Primary Associated Research Questions
Quantity	How many in the population are becoming affected, have become affected, and are now affected?
Location	Where in the population are affected individuals more or less likely to be found, with variation in occurrence and frequency differentiated by characteristics of time, place, and person?
Causes	What accounts for some individuals becoming affected whereas others are not?
Mechanisms	What are the underlying linked sequences of events and processes that account for the occurrence and for the persistence of the condition?
Prevention and control	What can be done to prevent occurrence of the condition, shorten its duration, or ameliorate its circumstances?

### Exhibit 3. Main Concepts, Research Designs, and Statistical Tools Associated With Each Rubric of Epidemiology

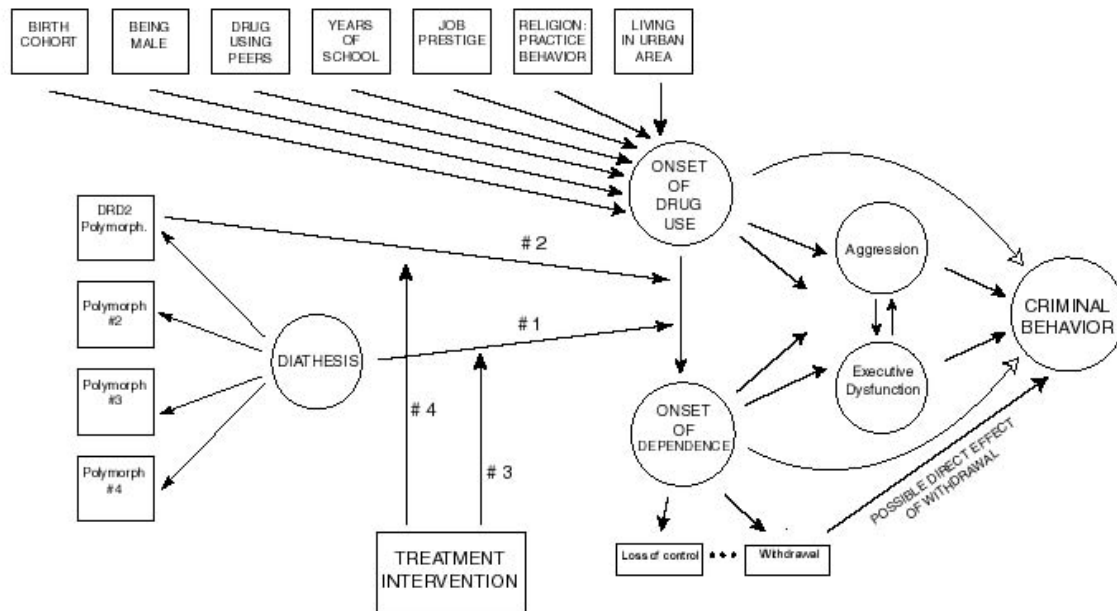
Main Rubrics	Illustrative Concepts	Main Associated Research Designs and Statistical Tools
Quantity	Point prevalence, interval prevalence, lifetime prevalence, and variance	Population census, observational ambidirectional or cross-sectional field survey, and variance estimation under complex designs
	Cumulative incidence and incidence density	Cohort and prospective study designs and multiwave panel study design
	Event rate, probability distributions, and density functions expectation	Vital statistics registration methods (birth, death), and rapid and continuing surveillance
Location	Prevalence correlate, factor, difference, ratio, odds ratio, and prev. = f (incidence, average duration); null hypothesis; statistical precision; likelihood principle; and tests of significance ( <i>p</i> -values statistical power)	Cross-sectional field studies, clinic-based and population-based case-control and case-base studies with prevalent (prevailing) cases; statistical measures of correlation and association; and univariate response regression models for description and prediction
	Incidence or risk correlate, risk factor, inverse risk factor, incidence difference, incidence rate ratio, cumulative incidence ratio, and incidence density ratio	All of the above, plus: Clinic-based and population-based case-control and case-cohort studies with incident (dynamically occurring) cases
Causes	Causal and preventive factors, Koch's postulates, criteria for evaluating causal significance of observed associations (e.g., dose-response relationships), counterfactuals, reciprocities, and effect-modification and interaction	All of the above, plus: Fine-grained and coarse-grained matching and stratification, direct and indirect adjustments, modeling with statistical adjustments, marginal and random effects models, hierarchical models (e.g., alternating logistic regressions), randomized trials, family history and twin studies, and instrumental variable models
Mechanisms	"Natural history" versus "clinical course" and mediation	All of the above, plus: Marginal and random effects longitudinal analysis models
Prevention and control	Efficacy versus effectiveness, preventive fraction, and attributable risk	Randomized controlled trial, and operations and systems research

**Exhibit 4. Prevalence of Drug Purchase Opportunity Among Youths  
12–24 Years Old, in Percent**



Source: Substance Abuse and Mental Health Services Administration, 1996 and 1997, *National Household Survey on Drug Abuse*, Rockville, MD: U.S. Department of Health and Human Services, Substance Abuse and Mental Health Services Administration.

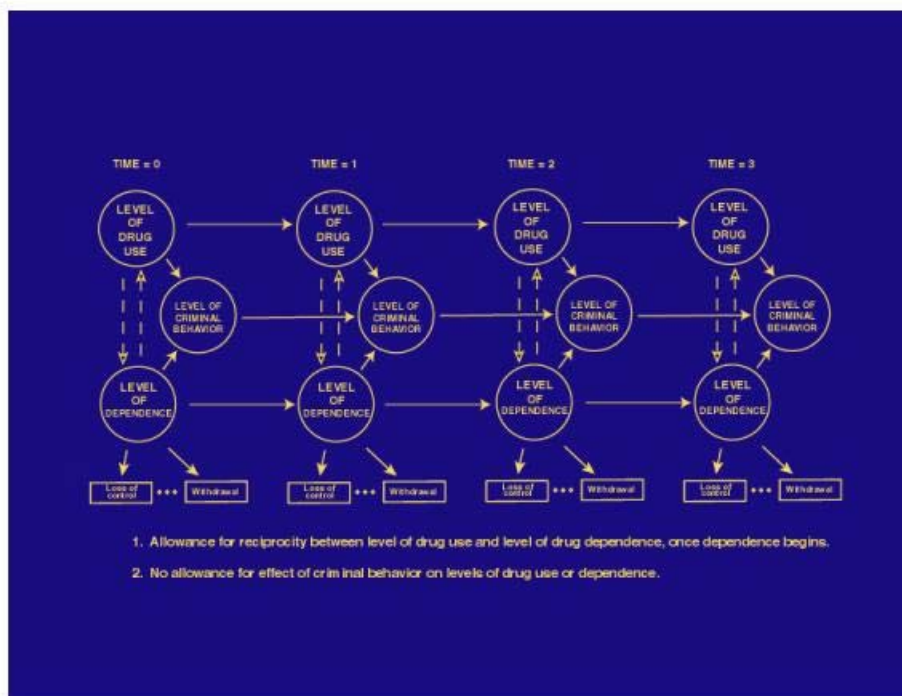
## Exhibit 5. Conceptual Model of the Influence of Drug Use and Drug Dependence on Criminal Behavior



Cartoon depiction of a mediational model linking a generic susceptibility trait (diathesis, path #1) with risk of making a transition from onset of drug use to onset of drug dependence and subsequent links to criminal behavior, directly and indirectly through drug-induced aggression and drug-induced disturbances in executive functions. Via path #3, treatment intervention might modify the expression of the generic diathesis (as manifest in covariation of multiple discrete polymorphisms) or might target a specific gene product or gene effect, with path #2 showing the putative gene effect and path #4 depicting the possibly specific effect of treatment intervention, over and above the intervention effect on the generic susceptibility trait.

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## Exhibit 6. Conceptual Model of the Drugs-Crime Relationship

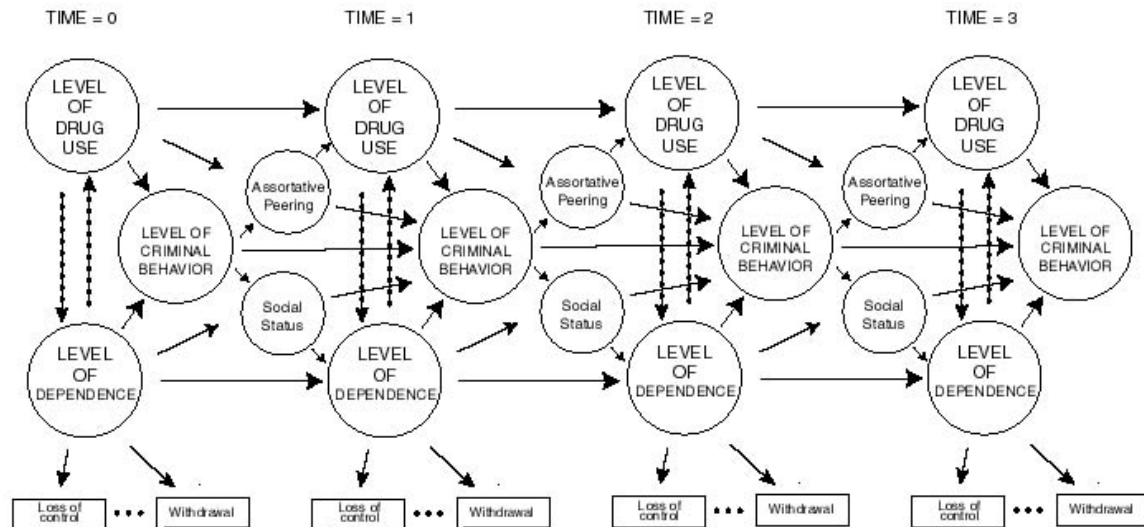


Notes: Depiction of a simplified longitudinal mediational model that links earlier levels of drug use and dependence to later levels of criminal behavior. For example, the level of drug use might produce intoxicating states that give rise to violent criminal behavior, even when the level of drug dependence is held constant (or kept at zero levels). Here, the level of drug dependence is tapped via a measurement model with clinical features of drug dependence, such as loss of control and withdrawal as the manifest indicators for levels of dependence. In this simplification, analogous measurement models for the level of drug use and the level of criminal behavior are not drawn but may be presumed.

In this depiction of the drugs-crime relationship, there is an allowance for reciprocity between levels of drug use and levels of drug dependence, once dependence begins. That is, there is not an acyclic dose-response relationship that links drug use to drug dependence. Rather, once the drug dependence process begins after first drug taking, the dependence process becomes a determinant of later levels of drug use. Most current conceptual models do not provide for this reciprocity.

This model is one that makes no allowance for the possible effect of criminal behavior on levels of drug use or dependence, but this defect is remedied in later elaborations of this model (e.g., see exhibit 7).

## Exhibit 7. How an Intervention Might Lead to a Change in the Drugs-Crime Relationship



Cartoon depiction of a longitudinal mediational model that links levels of drug use and levels of drug dependence with levels of criminal behavior. The model depicts criminal behavior's influence on subsequent levels of drug use via differential association as well as a possible influence on subsequent levels of drug dependence via changes in social status. Once the drug dependence process begins, there is a reciprocity, with level of dependence influencing level of drug use and vice versa. As in Figure 3, the level of drug dependence is manifest in the covariation of clinical features such as loss of control and withdrawal signs.

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**Exhibit 8. Criteria and Guidelines for Judging the Causal Significance of an Observed Association**

<b>Criteria/Guidelines</b>	<b>Associated Questions</b>
Temporal relationship	Is the temporal sequencing consistent with the idea that A causes B, or is there an ambiguity or the possibility that B causes A?
Biological or other theoretical plausibility	Is the idea that A causes B supported by theory or by trustworthy common experience and wisdom?
Consistency of the association	Is the available evidence consistent with the suspected causal link between A and B, or is there considerable inconsistency across studies?
Alternative explanations ruled out	If we are skeptical that A causes B or that B causes A, are there other specific alternative explanations for the observed statistical relationship between A and B, such as some background factor C that accounts for a spurious association between A and B?
Dose-response or gradient relationship	Is there regularity in the observed plot of B as a response to A? Where we see more of A, do we see more of B?
Strength of association	How strong is the relationship? Is it strong enough to make other alternative explanations less plausible?
Cessation effects	In this extension of the dose-response criterion, do levels of B drop substantially when A no longer is present?

## Exhibit 9. A Conceptual Framework for Research on the Drugs-Crime Relationship

